# THE PLANT DISEASE REPORTER

CROPS RESEARCH DIVISION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 45 Number 5

May 15, 1961

Growth Through Agricultural Progress

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THE PLANT DISEASE REPORTER

Crops Research Division, Plant Industry Station, Beltsville, Maryland
Volume 45, May 15, 1961, Number 5

Please note revisions in "Suggestions for Preparation of Manuscripts."

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# THE OCCURRENCE OF BLUE MOLD ON TOBACCO IN WEST GERMANY, SWITZERLAND, FRANCE AND OTHER EUROPEAN COUNTRIES

Furney A. Todd

## Summary

Blue mold has caused severe damage to tobacco in West Germany, France and Switzerland and, according to reports, in other countries including East Germany, Belgium, Holland, Austria, Poland, Czechoslovakia, Romania and to some extent in Italy. Damage to the 1960 crop has been estimated to be around $50,000,000 for all Europe. This disease has reduced yield as well as quality.

Environmental factors such as temperature, moisture, and overcast skies that exist in practically all countries where the disease has appeared are favorable for the causal agent of blue mold.

The disease may be controlled by a program that should extend from plant bed through harvest; however, success of this program will depend upon widescale grower acceptance throughout Europe.

Control will be expensive. The cost will range from $50 to $100 per acre, excluding labor.

The severity of the disease in 1960 and the cost of the preventive control program will undoubtedly discourage planting throughout Europe in 1961. There has been some decrease in acres planted each year in West Germany in the last 3 years. A sizeable reduction is expected for 1961, perhaps from 15 to 20%. This reduction will undoubtedly extend to other countries where blue mold has appeared.

The occurrence of blue mold in 1960 and the probability of its occurrence again in 1961 and for years to come will cause a reduction in total tobacco production for Europe. This will not only affect the pounds produced but may also affect leaf quality; therefore, this problem may greatly influence the sale of tobacco from the United States to European countries.

## INTRODUCTION

Blue mold, caused by Peronospora tabacina, was first discovered in Europe in October 1958 in a greenhouse in Great Britain. The origin of this infection is unknown. Then in 1959, blue mold was reported from the Netherlands and the northern part of West Germany in a few commercial fields of tobacco. Little damage was caused in 1959. However, the disease reappeared in 1960 throughout large areas of Europe with devastating results.

On June 24, 1960, blue mold was reported in the southern part of West Germany near Karlsrube. About 2 weeks later it was found throughout the cigar and burley-producing areas. The first occurrence in the flue-cured area near Bremen was on July 24. In addition, blue mold was also found in France, Belgium, Holland, East Germany, Switzerland, Czechoslovakia and Italy. Weather conditions were favorable in most of these countries and the disease reached epiphytotic proportions in July and early August. Losses in the countries of central and northern Europe were extensive. According to estimates, blue mold cost the farmers of Europe more than 50 millions of dollars in 1960. These losses were confined entirely to the field. No reports have been received of infections in plant beds in Europe.

Switzerland produces approximately 2500 acres of tobacco in ten different sections. Extensive damage occurred in the northern sections. In other parts of Switzerland only slight to moderate damage occurred. Growers were advised to harvest as rapidly as possible in order to avoid further damage.

Extensive damage in France was limited to the north where there was a 65 to 70% reduction in yield. Losses were much less in the middle of France and no blue mold was found in the south.

Growers in West Germany were more concerned about the blue mold disease than were the growers of France or Switzerland. At the request of the tobacco industry of West Germany, a study was made of the losses that occurred on burley, cigar and flue-cured tobacco.

## ESTIMATED LOSSES DUE TO BLUE MOLD IN WEST GERMANY IN 1960

Growers in West Germany planted approximately 16,500 acres of tobacco in 1960 in three different areas, separated by type.

Blue mold caused severe damage to all types. Many fields were observed where the loss approached 100%. Damage appeared to be more extensive in burley than in cigar or flue-cured (Virginia) type. Loss to blue mold in terms of reduced yield exceeded 50% for all types of tobacco. The estimated reduction in yield of burley was 67 to 70%; cigar, 50%; and Virginia type, 40 to 50%.

In addition to the reduction in yield, blue mold greatly reduced leaf quality. In most fields observed leaf area destroyed exceeded 50%. Since blue mold destroys mainly leaf lamina or leaf tissue, the leaves harvested from affected plants were of inferior quality.

Loss in Burley Tobacco: More than 50 tobacco fields were visited in the burley area around Heidelberg, Speyer, Neuforf and Mannheim. Blue mold was causing severe damage in all fields. Most severe damage occurred on the bottom leaves of the plants, but all leaves were affected. The disease was systemic in many plants, resulting in reduced growth.

In all fields visited the tobacco was spaced too close in the row to permit rapid drying of the early morning dew from the leaves. Wider spacing would be advisable since blue mold has become a problem.

Loss estimates ranged from 50 to 100% in the fields visited. Actual disease counts were made in a few fields to get some idea of extent of damage caused by the disease (Table 1).

Table 1. Estimated percentage leaf area destroyed in burley tobacco by the blue mold fungus.

| Field no. | Plant no. | % of leaf area destroyed by blue mold, leaf no. 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Average |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | 100 | 80 | 92 | 60 | 30 | 8 | | | | | 62 |
| | 2 | 90 | 85 | 50 | 15 | 12 | 45 | 10 | | | | 44 |
| | 3 | 90 | 95 | 80 | 95 | 98 | 25 | 40 | | | | 75 |
| 2 | 1 | 40 | 80 | 50 | 60 | 90 | 95 | 92 | 95 | 95 | | 77 |
| 3 | 1 | 35 | 55 | 80 | 95 | 80 | 30 | 25 | 90 | 50 | | 61 |
| 4 | 1 | 90 | 85 | 50 | 65 | 55 | 80 | 85 | 60 | 55 | | 70 |
| | 2 | 5 | 100 | 85 | 83 | 40 | 99 | 100 | 85 | 90 | 2 | 69 |
| | 3 | 95 | 85 | 75 | 65 | 80 | 40 | 95 | | | | 75 |

These counts were obtained by pulling all the remaining leaves on the plant and estimating the percentage of leaf area destroyed by blue mold. Damage to individual leaves on most plants checked was in excess of 50%, with some as high as 100%.

A leaf buyer also examined the leaves from Field No. 4 and rated them according to usefulness for the manufacture of smoking tobacco, cigarettes and/or cigars (Table 2). The two leaves acceptable for the manufacture of cigarettes and smoking tobacco had 5% or less of the area damaged. These same two leaves were rated as acceptable for the manufacture of cigars. In four other cases leaves with up to 55% damage were rated as usable but not desirable for the manufacture of cigars.

These data show that few tobacco leaves in the field at the time of this survey were usable for the manufacture of smoking tobacco, cigarettes or cigars.

Cured leaf of burley tobacco was examined in two barns. Leaves from these barns were selected at random and the percentage of diseased leaf area, as indicated by the cottony-like growth of the fungus, was estimated and recorded (Table 3).

Growers indicated that these leaves appeared healthy or free of blue mold when harvested and placed in the barn. It is believed that the leaves were infected before harvesting but symptoms had not yet appeared. Most likely sporophores and conidia were produced during the first 3 or 4 days in the curing barn.

A question was raised regarding the utility of tobacco on which damage occurred in the curing barn. The affected areas of the leaves remained intact during the curing process but were darker than normal where the mold-like growth was found on the underside. Many of the industry people felt that the presence of the fungus might affect the taste of the manufactured product. Others felt that these diseased areas would fall out or crumble during the redrying, fermentation and other manufacturing processes resulting in leaf of little value to the trade. There is no question but that the blue mold disease had lowered quality of the leaf in the two barns observed.

Loss in Cigar Tobacco: Only two fields were checked in detail in the cigar area. The first of the two fields was planted to Havana IIC. Damage was slight and ranged from 5 to 25% of the

Table 2. Acceptability of blue mold infected tobacco to trade.

| Plant no. | Leaf no. | % of leaf area destroyed by blue mold | General acceptability for manufacture of: smoking tobacco | cigarettes | cigars |
|---|---|---|---|---|---|
| 1 | 1 | 85 | not acceptable | not acceptable | not acceptable |
| | 2 | 85 | do. | do. | do. |
| | 3 | 50 | do. | do. | acceptable ? |
| | 4 | 65 | do. | do. | not acceptable |
| | 5 | 55 | do. | do. | do. |
| | 6 | 80 | do. | do. | do. |
| | 7 | 85 | do. | do. | do. |
| | 8 | 60 | do. | do. | do. |
| | 9 | 55 | do. | do. | acceptable ? |
| 2 | 1 | 5 | acceptable | acceptable | acceptable |
| | 2 | 100 | not acceptable | not acceptable | not acceptable |
| | 3 | 85 | do. | do. | do. |
| | 4 | 83 | do. | do. | do. |
| | 5 | 40 | do. | do. | acceptable ? |
| | 6 | 99 | do. | do. | not acceptable |
| | 7 | 100 | do. | do. | do. |
| | 8 | 85 | do. | do. | do. |
| | 9 | 90 | do. | do. | do. |
| | 10 | 2 | acceptable | acceptable | acceptable |
| 3 | 1 | 95 | not acceptable | not acceptable | not acceptable |
| | 2 | 85 | do. | do. | do. |
| | 3 | 75 | do. | do. | do. |
| | 4 | 65 | do. | do. | do. |
| | 5 | 80 | do. | do. | do. |
| | 6 | 40 | do. | do. | acceptable ? |
| | 7 | 85 | do. | do. | not acceptable |

Table 3. Estimated blue mold damage to cured leaves of burley tobacco.

| Leaf no. | % of leaf area with blue mold lesion: Barn 1 | Barn 2 |
|---|---|---|
| 1 | 65 | 35 |
| 2 | 40 | 55 |
| 3 | 80 | 80 |
| 4 | 50 | 95 |
| 5 | 60 | 80 |
| 6 | 90 | 30 |
| 7 | 95 | 25 |
| 8 | 92 | 90 |
| 9 | 90 | 66 |
| 10 | 95 | |
| Average | 76 | 61 |

leaf area destroyed. Less damage was observed in this field than in any other in West Germany, regardless of the type of tobacco. Blue mold was causing severe damage in the second field which was planted to the variety Geudertheimer (Fig. 1). All leaves on a given plant were affected with a high percentage of the leaf area destroyed. Field observations made while traveling through the cigar area indicated that blue mold was causing severe damage in most fields.

The first two primings of the cigar tobacco are used for wrappers and the remainder for filler. The tobacco obviously could not be used for wrappers; however, most of the crop possibly could be used for filler.

Only one barn was visited where cigar tobacco was being cured. Percentage of leaf area damaged by blue mold was estimated to be between 25 and 40. There again apparently the tobacco seemed healthy when placed in the barn, but the leaves were infected in the field and the re-

FIGURE 1. Blue mold damage to cigar tobacco in West Germany.

FIGURE 2. Blue mold damage to flue-cured tobacco in West Germany.

sulting damage occurred during the first 3 or 4 days of curing.

Loss in Flue-Cured Tobacco: Blue mold damage in the flue-cured area near Bremen was not so extensive as that caused to cigar or burley. Blue mold appeared in the flue-cured area about a month later than in the burley and cigar areas. Consequently, many of the growers were able to harvest 4 or 5 leaves per plant before the epiphytotic occurred. Even so, no tobacco was harvested in many fields that were planted late (Fig. 2). Also, the tobacco remain-

ing in most of the fields visited was damaged to the point that it was worthless. Systemic infection by the blue mold fungus was found in many fields.

The fields in the flue-cured tobacco-producing area are generally larger than those in burley or cigar areas. The tobacco was planted close; the rows were narrow and tobacco was spaced fairly close in the drill.

Information obtained from survey of the flue-cured area is presented in Table 4. The first field visited (Field 1) contained 11.2 acres. Most of the tobacco in this field had been harvested; in fact, only about five leaves remained on each plant. None of the tobacco that remained in the field was regarded as usable.

Table 4. Estimated percentage leaf area destroyed by the blue mold fungus.

| Field no. | Plant no. | % of leaf area destroyed by blue mold, leaf no. 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | Average |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | 80 | 65 | 85 | 95 | 85 | 60 | | | | | | | | | | | | 78 |
| | 2 | 65 | 55 | 30 | 30 | 75 | | | | | | | | | | | | | 51 |
| | 3 | 50 | 50 | 40 | 40 | 45 | | | | | | | | | | | | | 45 |
| | 4 | 40 | 45 | 40 | 60 | 75 | 15 | 10 | | | | | | | | | | | 45 |
| | 5 | 20 | 20 | 75 | 75 | 40 | 45 | | | | | | | | | | | | 48 |
| | 6 | 45 | 25 | 50 | 40 | 50 | 70 | 35 | 20 | | | | | | | | | | 42 |
| 2 | 1 | 100 | 100 | 100 | 100 | 85 | 90 | 80 | 85 | 95 | 75 | 90 | 85 | 80 | 85 | 90 | 95 | 85 | 89 |
| | 2 | 100 | 100 | 100 | 95 | 90 | 100 | 90 | 100 | 95 | 90 | 98 | 95 | 95 | 95 | 100 | 85 | | 96 |
| | 3 | 85 | 45 | 40 | 90 | 100 | 100 | 100 | 25 | 50 | 80 | 40 | 35 | 35 | | | | | 63 |
| 3 | 1 | 90 | 85 | 80 | 80 | 75 | 85 | 90 | 90 | | | | | | | | | | 84 |
| | 2 | 85 | 95 | 90 | 90 | 85 | 90 | 95 | | | | | | | | | | | 90 |
| | 3 | 100 | 100 | 85 | 85 | 85 | 70 | 100 | 60 | 70 | | | | | | | | | 84 |
| 4 | 1 | 100 | 100 | 100 | 100 | 100 | 80 | 80 | 80 | 60 | 60 | 75 | 85 | 75 | | | | | 85 |
| | 2 | 100 | 100 | 100 | 100 | 100 | 90 | 85 | 75 | 90 | 90 | 80 | 85 | 65 | 70 | 75 | 75 | | 86 |
| | 3 | 20 | 30 | 20 | 25 | 25 | 40 | 40 | | | | | | | | | | | 29 |
| 5 | 1 | 40 | 30 | 35 | 30 | 60 | 50 | 45 | 45 | | | | | | | | | | 42 |
| | 2 | 75 | 80 | 80 | 75 | 75 | 80 | 60 | 70 | 80 | 65 | | | | | | | | 81 |
| 6 | 1 | 80 | 80 | 75 | 75 | 25 | 30 | 90 | 75 | 75 | 60 | 75 | | | | | | | 70 |
| | 2 | 75 | 75 | 80 | 80 | 80 | 80 | 65 | 75 | 60 | 60 | 70 | 50 | 80 | | | | | 72 |
| | 3 | 95 | 60 | 75 | 75 | 60 | 60 | 60 | 70 | 60 | 75 | 80 | 75 | 75 | 60 | | | | 70 |
| 7 | 1 | 80 | 75 | 75 | 80 | 65 | 70 | 75 | 70 | | | | | | | | | | 74 |
| | 2 | 95 | 95 | 95 | 95 | 95 | 95 | 80 | 90 | 85 | 95 | | | | | | | | 92 |
| | 3 | 65 | 60 | 65 | 55 | 20 | 20 | 50 | 80 | 95 | 80 | 80 | | | | | | | 65 |
| 8 | 1 | 85 | 75 | 85 | 95 | 80 | 80 | 75 | 65 | 75 | | | | | | | | | 79 |
| | 2 | 30 | 20 | 35 | 40 | 30 | 25 | 35 | 35 | | | | | | | | | | 31 |
| | 3 | 25 | 25 | 60 | 75 | 80 | 75 | 80 | 60 | 75 | | | | | | | | | 70 |

Field 2, located on the same farm as Field 1, was planted late and little tobacco had been harvested. Damage from blue mold was so extensive that the tobacco remaining in the field at the time of the visit was not usable. Field 3 contained 3.7 acres and little tobacco had been harvested. Here, again, the loss was extensive. Field 4 was located on the same farm and no tobacco had been harvested from the field. Blue mold was very destructive, and completely destroyed the crop. Field 5 contained 23.7 acres. About four leaves had been removed from each plant before blue mold became severe. Again, the tobacco remaining in the field was almost worthless. Field 6 contained 10.7 acres and no tobacco had been harvested. The tobacco remaining in the field was almost completely destroyed by blue mold. From four to eight leaves had been harvested from each plant in Fields 7 and 8 and that remaining was worthless. Cured leaf was examined from one farm. Damage in this instance was slight since the tobacco was harvested before the disease became severe.

## EVALUATION OF THE PROBLEM IN EUROPE

Blue mold caused severe damage to all types of tobacco in West Germany in 1960. It is present throughout Switzerland and has caused severe damage in the northern area. The disease occurs in the northern and central tobacco-producing areas of France and has reduced the yield by as much as 75%. No reports of blue mold have been received from the southern area of France. The disease has been reported in East Germany, Belgium, Holland, Austria, Poland, Czechoslovakia and Italy, in addition to the countries mentioned above.

Environmental factors such as temperature, moisture, wind and sunshine greatly influence the activity of the blue mold fungus in tobacco (3). Each of these factors exerts its own effect and greatest damage occurs when all are combined in a manner conducive to disease development. Weather conditions are important from the standpoint of rapidity of spread and disease development.

Temperatures of the past growing season in West Germany were ideal for infection of tobacco by Peronospora tabacina, as well as for subsequent disease development and sporulation. Transplanting of tobacco to the field is usually nearly completed by May 15 in West Germany. Blue mold was first observed on June 24 in the southern area and on July 24 in the northern area. The temperature record for August was similar to the temperature record for July. The mean temperatures for the years 1881-1930, for June, July and August, are 63°, 68°, and 64° F, respectively (Fig. 3).

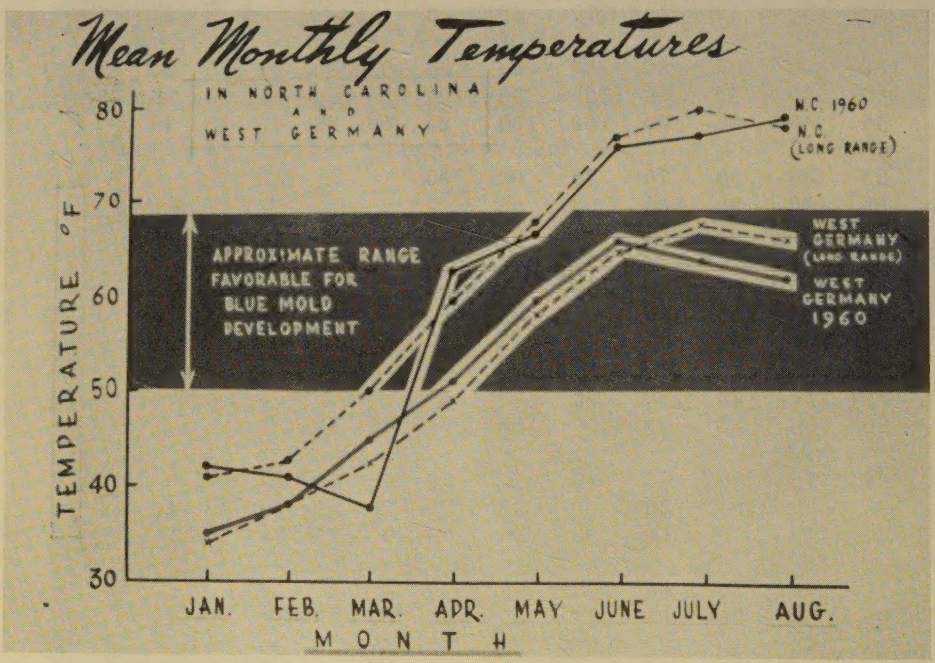


FIGURE 3. Mean monthly temperature -- North Carolina, West Germany.

Moisture is necessary for spore germination (2). In general, moisture was adequate for spore sporulation and germination during the months of June, July and August of 1960 in West Germany. The total precipitation for June and July and through August 23 was 1.8, 3.4 and 4.3 inches, respectively. Rains occurred almost every day during July and August and frequently during June. Reports from individuals indicate that heavy rains or downpours seldom occur in West Germany. The average rainfall for the period 1881-1930 for the months of May, June, July and August is 2.3, 2.8, 3.0 and 3.1 inches, respectively. If this amount of rainfall occurs as frequent showers, it would provide adequate moisture conditions for spore sporulation and germination.

The presence or absence of sunlight has pronounced effect on development of the blue mold disease. Direct exposure to sunlight kills the conidia in one hour (4). Without sun, they may live for a much longer period of time. Information on number of hours of sunshine per month indicates that many days in West Germany are characterized by overcast skies or cloudy

weather. This applies not only to the 1960 season but to previous seasons as indicated by the data from the period 1881-1930 for the months of May, June, July and August.

Wind is important from the standpoint of spread of conidia. Light winds are sufficient to carry the conidia for a distance of 75 miles (7). Light winds occur almost every day in West Germany during the months of May, June, July and August.

In summary, the mild temperatures, generally high relative humidity and frequent overcast skies are favorable for the growth of the causal agent as well as for the production, dissemination and germination of conidia. Consequently, the blue mold disease may seriously limit production in future years unless a vigorous control program is started immediately.

Unconfirmed reports indicate that weather conditions in Holland, Belgium, parts of France, Switzerland, Austria, Czechoslovakia, Poland and Romania are similar to the conditions that exist in West Germany. This would suggest that blue mold could be a serious problem in all of these countries unless a vigorous control program is started immediately. The widescale occurrence of blue mold in 1960 most likely will result in a large carryover of the oospores (1, 3, 5, 6, 7). The low temperatures that exist during the winter months may reduce this carryover to some extent; however, many investigators and research personnel in the United States feel that there will be sufficient carryover to start the disease in 1961.

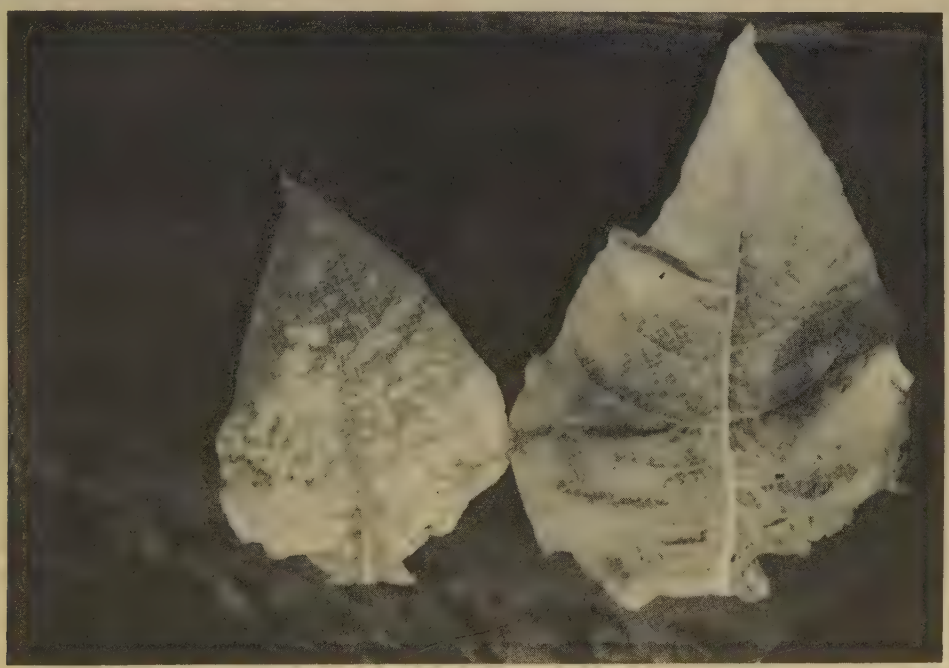

FIGURE 4. "Ray of hope for Europe." Left -- leaf from untreated plant. Right -- leaf from plant treated with maneb.

It is believed that it might be possible to control blue mold in Europe (Fig. 4). The control progr. n may be long and difficult. It must start in the plant bed and most likely have to continue through harvest. All known measures should be used including: 1) plant bed soil sterilization by heat or chemicals; 2) use of preventive fungicides such as manganese ethylene bisdithiocarbamate (maneb), and zinc ethylene bisdithiocarbamate (zineb) in the bed and field; 3) development of resistant varieties; 4) certain field management practices involving spacing, fertilization and rotation; 5) destruction of old crop residues to reduce carryover; and 6) elimination of perennial hosts in greenhouse.

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# PINE TWIST RUST (MELAMPSORA PINITORQUA) IN NORTH AMERICA[1]

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Abstract

Ponderosa pine (Pinus ponderosa) seedlings in a nursery of central British Columbia were found to be infected with pine twist rust, caused by Melampsora pinitorqua Rostr. The occurrence of the rust in North America and the susceptibility of ponderosa pine to M. pinitorqua are reported for the first time. The taxonomy, life history, etiology, and geographical distribution of the rust fungus are outlined.

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At the beginning of July 1960, a disease of ponderosa pine (Pinus ponderosa) in a forest tree nursery at Telkwa was brought to the author's attention. Telkwa is located in central British Columbia, approximately 400 miles northwest of the northern limit of naturally-occurring ponderosa pine. The pine was raised from seed planted in the spring of 1959 in a small bed that contained a few hundred seedlings only. At the time the disease was noted by Mr. J. T. Schmidt, Silviculture Supervisor, approximately one-quarter of the seedlings had already succumbed to it.

Examination of the seedlings by the author showed that the disease was pine twist rust, caused by Melampsora pinitorqua Rostr., a fungus well known as a parasite of young pines in Europe. A review of pertinent literature revealed that there are no records of ponderosa pine being susceptible to this disease, or of the disease ever having been observed on the North American continent. In view of the potential danger of foreign rust diseases to native trees, exemplified by the introduction of white pine blister rust (Cronartium ribicola J. C. Fisch.) to North America, nurserymen in the province were alerted, and the remainder of the ponderosa pine at Telkwa was destroyed.

## THE CAUSAL AGENT

Life History: "In spring, the basidiospores of Melampsora pinitorqua infect the young shoots of pine, forming a perennial, intercellular mycelium that grows in the bast, the medullary rays, and especially the phloem parenchyma. At the end of May or the beginning of June, pycnia develop on the newly infected young shoots, followed by caeomata[3] later on. The part of the twig around the rust scar becomes brown, resinous, and necrotic. As a result, small twigs die soon after the caeomata have developed; larger shoots survive but become twisted where the infection has taken place; hence the name Caeoma pinitorquum de Bary (1864)." (From Gaümann (6)).

In June and July the aeciospores are dispersed from the caeomata by the wind to infect the leaves of susceptible poplars. Approximately 2 weeks later, uredinia develop on conspicuous yellow spots of the infected poplar leaves. The urediniospores, in turn, cause additional infections on poplars and under favorable conditions the leaves may become heavily rusted by early fall. In the fall, telia develop in the infected, somewhat discolored poplar leaves, which are usually shed prematurely. The telia overwinter in the dead leaves on the ground. Basidia and basidiospores begin to germinate from the telia in the spring by the time the new shoots of pine begin to develop. The basidiospores once again infect the young shoots of pine, thus completing the 1- year life cycle of the rust fungus.

Under certain conditions and on certain hosts, Melampsora pinitorqua may overwinter in the buds of poplar as mycelium and then produce a crop of urediniospores on the petioles and young leaves in spring (12). Thus, in the absence of pine this parasite may persist as poplar leaf rust for unlimited periods, still capable of spreading the disease to pine but no longer requiring pine as alternate host for survival (12). On the other hand, in the absence of poplar the

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[1]Contribution No. 735 from the Forest Entomology and Pathology Branch, Department of Forestry, Ottawa, Canada. The co-operation of Mr. J. T. Schmidt, Silviculture Supervisor, British Columbia Forest Service, is gratefully acknowledged.

[2]Forest Entomology and Pathology Laboratory, Victoria, B. C.

[3]A caeoma (plural caeomata) is a spore-bearing structure of the rust fungi which lacks a covering membrane - an aecium without a peridium. See illustration in Cummins (4, p. 5).

fungus may persist as pine twist rust only until the infected pines have died. New infections of pine require the presence of poplars in the vicinity, whereas new infections of poplar do not require even the existence of pine.

Identification: M. pinitorqua belongs to a group of rust fungi "usually considered as species which do not differ essentially in morphological characters, but which possess different haplont hosts." Many modern uredinologists therefore follow Jørstad (8) in looking upon M. pinitorqua as a race of M. populnea (Pers.) Karst. rather than as a separate species, although the formal transfer from species to race has never been made. In its aecial state the rust can be recognized without difficulty for M. pinitorqua is the only fungus known to produce caeomata on pine.

Spore diameters of aeciospores from the Telkwa collections were slightly, but not significantly, greater than those described for the species (6, 7). Caeomata occurred not only on shoots but on cotyledons and young needles as well, confirming observations made by Garbowski on Scots pine seedlings in Poland (5). Mature aeciospores showed distinct bi-lateral thickening of the spore walls, indicating that the rust belongs to a species of Melampsora that bears its telia on poplars (15, p. 110). Neither the occurrence of caeomata on the needles nor the bilateral thickening of the aeciospore walls is noted by Gaümann, but otherwise the rust from Telkwa agrees well with M. pinitorqua as described in Gaümann's "Die Rostpilze Mitteleuropas" (6).

The specimens of infected ponderosa pine seedlings from Telkwa are deposited in the following herbaria: Victoria, B. C., Canada (DAVFP[4] 12177 and 12464); Ottawa, Canada (DAOM); and Lafayette, Ind., U. S. A. (PUR, the Arthur Herbarium).

Hosts and Distribution: Results of inoculation experiments have proved that the following tree species are susceptible to M. pinitorqua (6): for the pycnial and aecial states, Pinus mugo (mountain pine) and P. sylvestris (Scots pine); for the uredinial and telial states, Populus alba (white poplar), P. tremula (European aspen), and its hybrid, P. canescens. Host records unconfirmed by inoculation experiments include Pinus nigra vars., P. pinaster (13), and Populus canadensis (10).

Melampsora pinitorqua has been known to occur only in Europe, on the hosts listed above. No North American trees were known to be susceptible to the parasite before the discovery of the disease on ponderosa pine at Telkwa.

Damage and Control: Damage to pines becomes less as they get older. By the time they are 12 years old most trees are out of danger (2). Seedlings up to 3 years old may be killed (1) and saplings deformed with part of their shoots and leaders being killed or twisted. Adventitious budding around killed leaders may lead to "rosette" growth, hypertrophies, and similar abnormalities (2). Vigorous shoots may wall off rust-infected tissues by formation of wound periderm and thus survive the infection (11). The severity of damage to pine in Europe depends largely on weather conditions during the infection period and the proximity of susceptible poplars. Damage to poplar has been less severe than to pine. It is confined to the foliage, which yellows and may be shed prematurely. The resulting decrease in photosynthesis leads to a reduction in the annual growth increment.

Control of the pine rust can best be attained by either avoiding the cultivation of susceptible pines in the vicinity of susceptible poplars or by removing susceptible poplars from the vicinity of susceptible pines (2, 14). To protect pine nursery stock, spraying with a suitable fungicide may prove more practicable than eradication of susceptible poplars (3).

## DISCUSSION

The discovery of M. pinitorqua at Telkwa raises a number of questions, among them: (a) How reliable is the identification of the parasite as M. pinitorqua? (b) Has M. pinitorqua been introduced to North America by man? (c) If so, how, where, and when has it been introduced, and what is its actual present geographical distribution? (d) What species of North American trees besides ponderosa pine are potential hosts of the parasite?

To confirm the identity of the rust and to test its host relationship, attempts were made to infect trembling aspen (Populus tremuloides) and white poplar. Unfortunately the aeciospores used as inoculum were partly germinated and no longer viable when they arrived from Telkwa. No infection was obtained. The facts alone that the rust is a Caeoma on "hard" (2- or 3- needled -) pine, that its aeciospores show bi-lateral thickening (see section "Identification"), and that it agrees closely in host symptoms as well as in pycnial and aecial morphology with M. pinitorqua can, however, be considered as sufficient evidence for its identity.

The recent discovery of M. pinitorqua does not of necessity, as one might be led to believe,

[4]For abbreviations of names of herbaria Lanjouw and Stafleu (9) are followed.

imply its recent introduction from Europe. In an attempt to trace the origin of the rust, a search was made in the vicinity of the nursery for alternate hosts from which, undoubtedly, the disease must have spread to pine. It revealed one small tree of white poplar, the only poplar found that is known to be susceptible to M. pinitorqua. This tree was obtained as a cutting in the Abbotsford area near Vancouver, British Columbia; planted and raised in a nursery at Terrace, approximately 60 miles west of Telkwa; and finally transplanted to the nursery at Telkwa 30 feet from the seedbed which later became infected with M. pinitorqua. The tree bore green foliage and was approximately 2 feet high when it was planted at Telkwa in October 1959. No rust was found on its leaves in 1960; nevertheless the author believes that the rust may have been introduced with this tree to Telkwa, not from Europe, but from either Abbotsford or Terrace, British Columbia. A search for the rust at these locations will be made.

In view of the distribution of M. populnea, restricted in North America to regions with mild climate, and of the fact that M. pinitorqua is unrecognizable as such in its uredinial state, it seems possible that the so-called pine twist rust has existed in North America as a leaf rust of white poplar, under the name M. populnea, ever since white poplar became widely cultivated on this continent. The prevalence and potential danger of pine twist rust to forestry in North America cannot be fully assessed until inoculation experiments designed to elucidate the taxonomy and host relationship of M. populnea have been carried out.

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# ATTEMPTS TO CONTROL DISSEMINATION OF INTERNAL CORK VIRUS OF SWEETPOTATOES WITH INSECTICIDES

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## Summary

Experiments were conducted in North Carolina for 4 years to determine the effectiveness of insecticides to reduce the dissemination of internal cork virus (ICV) in sweetpotatoes. Experiments were designed to prevent immigrating viruliferous insects from inoculating virus-free plants, and to prevent insects from acquiring and spreading the virus within a planting. DDT, parathion, demeton, Perthane, methoxychlor plus malathion, and toxaphene plus malathion were applied to the foliage and aldrin and heptachlor as soil treatments. Foliage treatments were made at weekly intervals (demeton, 21 day intervals) throughout the growing seasons. No reduction in virus dissemination was obtained with any of the insecticide treatments. The failure to obtain some reduction in the acquisition and spread of virus within artificially infected plantings indicated that ICV is a non-persistent virus.

The natural spread of the internal cork virus (ICV) of sweetpotatoes led earlier investigators to postulate that the virus was insect borne. Later, Myzus persicae (3, 10), Macrosiphum solanifolii (3), and Aphis gossypii (4) were reported as vectors of ICV. These observations and findings suggested that virus dissemination might be reduced or even eliminated by applying an appropriate insecticide. Prior to the discovery of vectors, Nusbaum (8) tested DDT, and Rankin (9) tested several insecticides but both failed to prevent dissemination of internal cork virus from infected to virus-free plantings. Recently, Kantack, et al. (5) reported the results from tests extending over five growing seasons. During one growing season (1955), they obtained a reduction in virus dissemination with bi-weekly applications of 10% DDT throughout the season and 2 pounds/acre of O,O-diethyl S-isopropylthiomethyl phosphorodithioate (American Cyanamid systemic compound 12008) from mid-June to mid-July. However, in later tests they obtained no reduction in virus dissemination with these or other insecticides.

The objectives of this investigation were to determine whether dissemination of the virus could be reduced or eliminated by insecticides, and to gain information on the nature and habitat of the vector or vectors. The insecticide studies were conducted during four growing seasons.

## MATERIALS AND METHODS

Two types of experimental plantings were made. In the first, virus-free plants were set adjacent to infected sweetpotatoes and the virus-free plants were treated with insecticides. In this case it was assumed that viruliferous insects could move directly from the infected plants to the virus-free plants and immediately inoculate the latter. Under these conditions an insecticide would have to paralyze or kill the insect rather quickly to prevent virus transmission. In the first test (1953), a replicated split-plot design was employed in which soil and foliage insecticides were tested. The plots were parallel with an infected planting and the 12-plot rows extended 50 feet from the latter. The soil insecticides 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-endo-exo-5,8-dimethanonaphthalene (aldrin), 3a,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene (heptachlor), and no treatment were the major plots and a dust mixture of O,O-dimethyl dithiophosphate of diethyl mercaptosuccinate (5% malathion) plus 1,1,1-trichloro-2,2-bis(p-methoxyphenyl) ethane (10% methoxychlor) and no treatment were the subplots. The major plots contained four rows of plants 40 feet long and were replicated four times. The subplots were two rows each. The soil insecticides were applied as emulsions at the rate of 6 pounds active/acre and disked into the soil within 24 hours after application. Virus-free plants were set 3 days after making the soil treatments and the weekly foliage treatments were started 2 weeks after transplanting. The foliage insecticide part of this experiment was repeated in 1958. In this case, duplicate plots of four rows 50 feet long were parallel with the infected planting and separated by 25 feet of fallow soil. One plot received weekly applications of insecticides that were started the day the plants were set.

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The second type of planting was designed to test the efficacy of foliage insecticides in preventing the acquisition and spread of ICV within a sweetpotato planting. These plantings were made some distance (350 to 2500 feet) from other sweetpotatoes infected with the virus, and contained both infected (10%) and virus-free plants (90%). All infected plants were indentified with stakes. Both the infected and virus-free plants were treated with insecticides. It was postulated that the vector would have to enter the planting and acquire the virus from infected plants before it could inoculate the virus-free plants, thus giving the insecticides more time to act upon the insect. At harvest, the originally infected plants and their roots were removed before the remaining plants were lifted. Tests of this kind, involving replicated and single plot plantings, were conducted during three seasons. Each season different insecticides were employed with appropriate controls. These details will be given with the results.

The same virus-free Porto Rico stock was used in all tests. This stock was kept free of the virus by growing it in isolation. The experimental plantings were made between June 7 and July 2 for the several years and the cultural practices were those employed in commercial production. The roots were harvested in October or November, cured, and stored at warm temperatures for 5 to 6 months and then they were sliced and examined for internal necrosis. The efficacy of the insecticide treatments is based upon the percentage of roots from treated and nontreated plants that had internal necrosis.

All insecticidal dusts and sprays were applied with hand-operated dusters and sprayers. Treatments were started at planting or soon after and continued at approximately weekly intervals until harvest. The foliage dusts were applied at approximately 30 pounds/acre when the vines covered the ground, and the sprays were applied until the foliage was wetted.

## RESULTS

Protection of Virus-free Plants from Viruliferous Vectors: In the split-plot test involving soil and foliage insecticides applied to virus-free plants, there was no evidence of reduced virus dissemination due to insecticide treatments (Table 1). The proportion of roots having internal necrosis was relatively large and similar for all treatments. One could hardly expect the

Table 1. Incidence of internal cork symptoms in roots of virus-free sweetpotato plants grown adjacent to infected plants and treated with soil and foliage insecticides.

| Insecticide and dosage | % roots with internal necrosis |
|---|---|
| Soil treatments: | |
| Aldrin, 6 pounds/acre | 78.2 |
| Heptachlor, 6 pounds/acre | 83.2 |
| Soil and foliage[a] treatments: | |
| Aldrin, 6 pounds/acre plus dust | 72.6 |
| Heptachlor, 6 pounds/acre plus dust | 76.9 |
| Foliage treatment: | |
| Malathion + methoxychlor, 30 pounds/acre | 81.9 |
| No treatment | |
| None | 79.7 |

[a] Foliage dust mixture contained 5% malathion plus 10% methoxychlor applied weekly.

non-systemic soil insecticides to destroy airborne vectors. These insecticides were included to see if a soilborne vector may be involved in virus transmission.

In the 1958 test with duplicate plots of virus-free plants, the treated plot received an insecticide treatment immediately after planting and a total of 17 applications during the growing season. The first three dust applications were 4% malathion and thereafter a dust mixture of chlorinated camphene containing 67-69% chlorine (10% toxaphene) plus 3% malathion. The percentages of roots with internal necrosis from the treated and nontreated plots were 5.2 and 6.0, respectively. Again, there was no evidence of reducing virus dissemination with the insecticide treatments. The failure to prevent viruliferous insects from inoculating disease-free sweetpotatoes with ICV is in keeping with results from similar experiments with other viruses (1).

Preventing Virus Acquisition and Spread: The first mixed planting (1951) of infected and virus-free plants was a replicated test 350 feet from other sweetpotatoes infected with internal cork virus. There were four treatments: O,O-diethyl O-p-nitrophenyl phosphorothioate (para-

thion), dichloro diphenyl trichloroethane (DDT), O,O-diethyl O(and S)-2-(ethylthio)ethyl phosphorothioate (demeton), and no insecticide in randomized blocks separated by 10-foot borders. Each plot consisted of three rows 40 feet long. The plots within blocks were separated by a nonplanted row. The first application of insecticides was made immediately after planting and continued at weekly or tri-weekly intervals, depending on the insecticide used. The incidence of internal necrosis in roots from plants treated with insecticide regimes is summarized in Table 2.

No reduction in the dissemination of ICV was obtained with any of the insecticides. Roots from plants receiving the insecticide treatments had approximately the same proportion exhibiting internal necrosis as did roots from plants receiving no insecticides.

Table 2. Dissemination of internal cork virus within an infected planting of sweetpotatoes treated with insecticides[a].

| Insecticide | Number of applications | % roots with symptoms |
|---|---|---|
| Parathion-dust 1 1/2% | 20 | 36.5 |
| DDT - dust 5% | 19 | 31.8 |
| Demeton-spray 1:2000 | 6[b] | 39.1 |
| None | | 39.5 |
| L.S.D. | | N.S. |

[a] 10% of original plants infected. These were removed and discarded before harvest.
[b] Two applications of parathion at 7-day intervals, then demeton at 3-week intervals.

The failure of insecticides to reduce the dissemination of ICV in this experiment suggests that the vector is capable of acquiring and disseminating the virus in a relatively short time. Although this planting was 350 feet from the nearest sweetpotatoes infected with ICV, later experiments at this station and in Louisiana (2, 7) have shown that some of the vectors entering this planting may have carried virus over this distance. However, the distribution of infection in the planting suggested that the source of vectors was from another direction. There was a significant difference in the incidence of dissemination from one end of the plot area to the other (a distance of 200 feet). Roots from a block of plots adjacent to a soybean field had an average of 49% with internal necrosis, while those from the most distant block had an average of 29.3%. This difference in dissemination of the virus down the field suggested that the vectors were coming from the planting of soybeans.

The ability of insects to acquire and disseminate ICV within a planting treated with insecticides was further tested in 1957 and 1958. Each year duplicate plantings were established some distance (1500 feet in 1957 and 2500 feet in 1958) from other sweetpotatoes. In addition, the duplicate plantings were about 100 yards from each other in 1957, and 2500 feet in 1958. Each planting consisted of 200 plants set in four rows 50 feet long. One planting received insecticides applied as a dust at weekly intervals and the other planting served as the untreated control. In 1957 the first four dust applications were 4% malathion and the 10 succeeding applications were 1,1-dichloro-2,2-bis(p-ethylphenyl)ethane (5% Perthane). The percentages of roots with internal necrosis were 54.9 for the treated planting and 41.3 for the untreated. In the 1958 test the first three dust applications were 4% malathion and the last 13 were 10% toxaphene plus 3% malathion. In this test, the percentages of affected roots were 0.4 and 0.2, respectively, for the treated and untreated plantings. Virus dissemination was very low in 1958 in comparison with 1957. This difference in virus dissemination was evident in other experiments conducted these 2 years (2). In neither test did the insecticide treatments reduce virus dissemination within the plantings. In fact, the data suggest that the insecticide treatments increased virus dissemination within the treated planting. In both years, roots from the treated plantings had a larger proportion with internal necrosis. Unfortunately, the data were not susceptible to statistical analysis. Other instances of increased virus dissemination following the application of insecticides have been noted (1).

## DISCUSSION

The use of insecticides either as soil treatments, foliage treatments, or both, failed to reduce the dissemination of ICV. This was true whether the insecticides were used to protect virus-free plantings from invading viruliferous insects or to prevent the acquisition and spread of virus within a planting. The failure to reduce virus dissemination when the treated virus-free plantings were adjacent to a nontreated infected crop is not surprising since viruliferous insects could move to the virus-free plants and inoculate them before being paralyzed or killed by the insecticide. The one case of reduction in ICV dissemination reported by Kantack, et al.

(5) was also an experiment in which virus-free plants were protected with twice weekly applications of insecticide from immigrating viruliferous insects. They infer that the success obtained in 1955 was associated with greater vector activity. The proportion of roots with internal necrosis in tests summarized in Tables 1 and 2 also indicate the presence of many vectors during these growing seasons, but no benefit was derived from weekly foliage insecticide treatments.

The ineffectiveness of insecticides in reducing the acquisition and dissemination of virus within plantings indicates that ICV is a non-persistent virus. The pattern of ICV dissemination from a source of inoculum also indicated to Martin and Kantack that it is a non-persistent virus (6). After reviewing the results of many reports on the use of insecticides to control virus dissemination, Broadbent (1) concludes that little success has been achieved with insecticides in reducing dissemination of non-persistent viruses within plantings; while good success has resulted with various persistent viruses.

The failure to obtain control may be related also to the size of the plot areas. Had the artificially infected plantings been several hundred feet in either dimension, some control may have been achieved toward the center of the planting where a constant replacement by large numbers of immigrant vectors may have been reduced. The plots employed were small and alatae vectors from surrounding nontreated crops or plants had to move only short distances to reach the centers of treated plots.

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# NATURAL OCCURRENCE OF HOJA BLANCA ON WHEAT AND OATS[1]

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Hoja blanca, an insect-transmitted virus disease of rice, was found in 1960 for the first time occurring naturally on wheat and oats in experimental nurseries at the Palmira Agricultural Experiment Station in the Department of Valle del Cauca, Colombia. Leaf striping and chlorosis and inflorescence sterility, typical of the symptoms of the disease on rice[2], were observed. A rice nursery adjacent to one of the wheat fields was heavily infected by the virus. The severity of attack on the wheat followed an obvious gradient, with the greatest number of diseased plants found nearest the rice.

Sogata orizicola Muir, the principal vector in rice, has given positive results in cage studies of transmission of the virus from rice to wheat, to oats, and to barley as well as to other species of the Gramineae. S. cubana (Crawf.), the vector for Echinochloa colonum, does not transmit the virus from rice to rice or from rice to wheat, to oats, or to barley[3]. Both species of the insect were collected in large numbers from the rice nursery, but it is not known with surety which was involved in the field infection of wheat and oats.

Counts of diseased and healthy plants were taken in both the United States Department of Agriculture International Oat Rust Nursery and the 13A Puerto Rico Oat Rust Nursery. Approximately one-third of all the lines had some symptoms of hoja blanca. The following had more than 25% infected plants:

| 1959 International Oat Rust Nursery | |
|---|---|
| Ukraine | C.I. 7007 |
| Avena abyssinica | C.I. 7233 |
| Newton | C.I. 6642 |
| Minhafer | C.I. 6913 |
| Tennex x (Victoria x Hajira-Banner) | C.I. 6994 |
| Sel. D.L.M. 3 Ins. S.F. | C.I. 7055 |
| Sel. 189 Ins. S.F. | C.I. 7056 |
| New Nortex x Landhafer | C.I. 6998 |
| (H-J x (Fulwin x Lee-Victoria)) x (Bond-Anth.-Land.-Colo) | C.I. 7401 |
| Sel. D.L. 41372 | C.I. 7171 |
| D.L.M. 3 | C.I. 7172 |
| (Landhafer-(Mindo x H-J)-Andrew) x Branch | Bulk line Sel. 15-2 |
| (Landhafer-(Mindo x H-J)-Andrew) x Roxton | Bulk line Sel. 54-2 |
| **1959 Puerto Rico Oat Rust Nursery (13A)** | |
| Awnless Culred | C.I. 2676 |
| Black Bell | C.I. 1767 |
| Bombo | C.I. 2685 |
| (Bond-Iogold) x (Victoria x Hajira-Banner) | C.I. 5907 |

From a wheat crossing block of 294 varieties and lines representing a broad spectrum of germ plasm, the following had a high incidence of infection with hoja blanca:

| | |
|---|---|
| Triticum sphaerococcum | P.I. 168685 |
| USSR No. 23824 (T. sphaerococcum) | P.I. 115818 |
| Timstein x Newthatch | R.F. 3770 |
| ND4 x Lee | N.D. 90-3 |
| Willet x Norin 10-Baart | II-7033-2c-4h-1r-3m |
| T. durum | C.I. 7315 |

In addition to the above, several hybrids from a wheat breeding nursery, involving crosses with T. vulgare and T. durum, were rated as susceptible.

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[1]Paper No. 135 of the Agricultural Journal Series of the Rockefeller Foundation.

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# EVALUATION OF AN EXPERIMENTAL NEMATICIDE O,O-DIETHYL O-2-PYRAZINYL PHOSPHOROTHIOATE[1]

R. E. Motsinger

## Abstract

The organic phosphate compound O, O-diethyl O-2-pyrazinyl phosphorothioate (Cynem - American Cyanamid 18133) was tested for nematicidal activity toward Meloidogyne incognita acrita in greenhouse and laboratory tests. In greenhouse tests the nematicide was mixed into the top 4 inches of nematode-infested soil in 2-gallon crocks 2 weeks and 1 week before planting, at planting, and 10 days after planting tobacco seedlings. Excellent nematode control was obtained at both 10- and 20-pounds per acre with all times of treatment. Seedlings in all treated soil were stunted for the first 2 weeks after planting and necrotic lesions appeared on the leaves, except on plants receiving the postplant treatments, but the plants had recovered and were equal in size to the controls 38 days after planting. Shoots of tobacco plants growing in treated soil were toxic to aphids (Myzus sp.) feeding upon them, indicating that the nematicide was absorbed by the roots and translocated throughout the shoot. The chemical exhibited long residual nematicidal activity in the soil; nematodes were effectively controlled in infested soil for at least 12 weeks after treatments. In laboratory tests, larvae and eggs incubated for 24 and 48 hours, respectively, in various concentrations of chemical in water solutions, then washed and placed around roots of tomato seedlings growing in soils, survived and caused root galling only if concentrations of less than 1000 ppm of chemical were used. No eggs hatched while egg masses were immersed in solutions of this chemical even at the lowest concentration used, 0.1 ppm. An inverse relationship existed between the number of galls on tomato roots and the concentration of chemical in which the egg masses had been incubated.

## INTRODUCTION

The expanding use of nematicides is making possible economic production of susceptible crops in an increasing acreage of nematode-infested fields. The search continues for nematicides which will be more effective, have low phytotoxicity, will be easier, safer and simpler to apply, and have long residual nematicidal activity in the soil. Several reviews summarize past work on chemical control of nematodes (9, 12, 13).

Several organic phosphate compounds have been tested for nematicidal activity. One of the first commercially available compounds was O-2, 4-dichlorophenyl-O, O-diethyl phosphorothioate (VC-13). This material had fairly low phytotoxicity, but was not as nematicidal as the halogenated hydrocarbons. It was reported that this compound applied as a drench to infested soil reduced the population of plant-parasitic nematodes and had long-lasting residual toxicity against both nematodes and insects (4).

O, O-diethyl-(2-ethylmercaptoethyl) thiophosphate (demeton) (Systox) applied as a 0.1% water drench to soil infested with root-knot nematodes (Meloidogyne incognita acrita Chitwood) reduced, but did not eliminate, infection of cucumber seedlings grown from seed planted immediately after soil treatment; applied as a 0.5% drench the material was phytotoxic and nematode control was only slightly improved (11). A few nematodes developed to maturity in the roots of seedlings grown in infested soil treated with the 0.5% drench, and eggs from masses extruded by mature females were viable when placed in water. Egg hatch was inhibited in solutions of 0.05% of demeton, but when the eggs were washed and placed in distilled water they hatched readily. Sprays and drenches with demeton were used to control the stem and bulb nematode, Ditylenchus dipsaci Kuehn, infecting alfalfa (2).

Other organic phosphates generally used as insecticides have found limited application against certain nematodes. O, O-diethyl O-p-nitrophenyl phosphorothioate (parathion) sprayed on infected strawberry plants controlled foliar nematodes, Aphelenchoides fragariae (Ritzema Bos) Christie, according to Raski and Allen (10). Control of Aphelenchoides ritzema-bosi

[1]Contribution No. 3215, Scientific Article No. A894, of the Maryland Agricultural Experiment Station, Department of Botany. Adapted from a thesis submitted to the Graduate School for the degree of Master of Science. Sincere appreciation to Drs. W. R. Jenkins and O. D. Morgan for research guidance and to Dr. L. R. Krusberg for guidance in thesis preparation.

(Schwartz) Steiner on infected chrysanthemums with parathion sprays has also been reported (3, 5). Root-knot nematodes were controlled on tomato when parathion was mixed thoroughly with nematode-infested soil (6).

Barker and Sasser (1) reported control of Ditylenchus dipsaci on alfalfa by mixing Cynem with nematode-infested soil prior to planting in greenhouse tests.

## MATERIALS AND METHODS

The methods used to evaluate Cynem were a modification of those discussed by McBeth and Bergeson (8). The chemical in water solution was tested in vitro for toxicity to eggs and larvae of M. incognita acrita, and was applied in a 5% granular formulation to soil in greenhouse tests. All rates of application refer to the quantity of technical chemical applied and not the amount of formulation.

Greenhouse Tests: Phytotoxicity to tobacco and nematicidal activity against root-knot nematodes was studied using the granular formulation of Cynem. The chemical was thoroughly mixed into the top 4 inches of fine sandy loam soil contained in 2-gallon crocks. The amounts of the chemical applied to the soil were based on the fraction of the surface area of an acre in the surface area of a crock. Rates of 10 and 20 pounds per acre were used in all greenhouse tests.

In the phytotoxicity and nematicidal activity tests, both rates of Cynem were applied to the soil in the crocks 2 weeks and 1 week before planting, immediately before planting, and as a sidedress treatment 10 days after planting. In the sidedress treatment the chemical was placed 1.5 inches deep in the soil in bands on both sides and 2 inches from bases of the plants. Soil in all crocks treated with the chemical was inoculated with equal amounts of finely chopped roots of tomato heavily galled by M. incognita acrita. Soil in three of the control crocks was inoculated with the galled tomato-root inoculum and soil in the remaining three crocks was not inoculated. Three crocks received each treatment, and two Catterton variety tobacco seedlings were planted in each. The seedlings, 3 weeks old when transplanted to the crocks, were harvested 38 days after transplanting. Top, root and total weights, and root-knot indices were recorded. Galls that developed on roots of plants receiving the sidedress treatment were placed in soil around tomato seedlings as a bioassay for the presence of viable larvae.

The residual nematicidal activity of Cynem was also tested, again at rates of 10 and 20 pounds of the chemical per acre. At 2-week intervals up to a total of 12 weeks after the chemical was applied to soil in crocks, root-knot nematode inoculum consisting of finely chopped heavily-galled tomato roots was mixed into the soil in four crocks. Approximately the same amount of inoculum was added to each crock. Two more crocks lacking chemical were inoculated with nematodes as controls. Three 3-week-old tomato seedlings, variety Chesapeake, were planted in each crock 1 week after nematode inoculation. Plants were harvested 27 days after transplanting to the crocks and the roots were indexed for root-knot galling on the basis of 1 = no galls, 2 = 1-25% galled roots, 3 = 26-50% galled roots, 4 = 51-75% galled roots, and 5 = 76-100% galled roots.

Laboratory Tests: The effect of Cynem on egg masses and larvae of M. incognita acrita was tested in vitro. Five egg masses from roots of infected tomato plants were placed in dishes containing 10 ml of each of the following concentrations of this chemical in distilled water: 0, 200, 400, 800, 1000, 5000, and 10,000 ppm. Larvae, 100 per dish, were treated similarly to the egg masses except the concentrations of Cynem used were: 0, 1, 10, 100, and 1000 ppm. Each concentration was replicated three times. Egg masses were incubated in these solutions for 48 hours and larvae for 24 hours at 25°C. Egg masses were checked at the end of 48 hours for the number of larvae hatched in the different concentrations of chemical. Then both egg masses and larvae were washed thoroughly with distilled water and placed 1.5 inches deep in the soil around tomato seedlings growing in 4-inch pots. After 30 days the root systems were washed free of soil and rated for galling as previously described.

## RESULTS

Greenhouse Tests: Phytotoxicity of Cynem to tobacco was obvious 1 week after transplanting seedlings to treated soil regardless of time or rate of application of chemical. Seedlings growing in treated soil developed small, round, necrotic lesions on the lower leaves within 1 week after transplanting and were also stunted as compared with seedlings in untreated soil. Both necrotic lesions and stunting were more severe in tobacco seedlings transplanted into soil immediately following application of Cynem than in those planted 2 weeks after treatment. No

necrotic lesions developed on any plants grown in untreated soil. Necrotic lesions did not develop on leaves of tobacco plants receiving the sidedress chemical treatment, but the plants were slightly stunted. All plants in treated soil were about equal in size to those in nematode-free untreated soil when harvested and no phytotoxic symptoms remained.

Cynem gave excellent nematode control in all treatments. Root-knot indices were significantly lower in root systems from plants grown in treated than untreated soil at the 1% level of significance using Duncan's range test (7). There were no galls on the roots of plants grown in soil treated at either rate of the preplant or immediately before transplanting treatments of Cynem and only a few galls occurred on roots of plants to which the sidedress treatments were applied. When egg masses from the plants receiving the sidedress treatment were placed in soil planted with tomato seedlings the tomato roots became galled in all cases. Dry weights of roots, tops and total plant were unaffected by chemical treatment of the soil or inoculation with root-knot nematodes.

Green peach aphids (Myzus sp.) were killed when they fed on tobacco plants growing in soil treated with Cynem. Aphids occurred in large numbers on plants growing in soil with or without root-knot nematodes, but not treated with chemical. All methods of application of the material were equally effective in keeping plants free of aphids. No living aphids were found on leaves of plants growing in treated soil where leaves of control plants, heavily infested with aphids, overlapped leaves of plants in treated soil.

In the test of residual nematicidal activity of Cynem in the soil, complete control of root-knot nematodes was obtained in all inoculations up through 12 weeks after treatment (Fig. 1), but phytotoxic symptoms appeared on the tomato plants. The symptoms on tomato were stunting and a marginal burn of the lower leaves. Tomato plants were stunted at both rates of nematicide application through the planting 6 weeks after treatment. Stunting was more severe at the higher rates of chemical and with the shorter waiting periods.

Laboratory Tests: In vitro tests indicated that a solution of 1000 ppm of Cynem was lethal to both larvae and eggs of M. incognita acrita when immersed for 24 and 48 hours respectively (Table 1, Fig. 2). No eggs hatched while they were immersed in even the lowest concentration of Cynem tested, but galls were formed on tomato roots when treated egg masses were washed with distilled water and placed in the soil around tomato plants except those exposed to concentrations above 800 ppm. Eggs hatched readily in the distilled water controls. Exposure of egg masses to the lowest concentration of chemical tested, 200 ppm, reduced the number of galls on the tomato bioassay plants 40% as compared with the controls. Higher concentrations further reduced galling of test tomato plants and egg masses subjected to 1000 ppm or above concentration solutions of this material no longer caused gall formation on tomato roots.

Larvae placed in solutions of Cynem were inactivated but not killed, except at the highest concentration used, 1000 ppm. The inhibition of nematode movement was inversely correlated with the concentration of the chemical in which the larvae were immersed (Table 1). When treated larvae were washed in distilled water and placed in the soil around tomato seedlings, galling was similar on the roots of all plants inoculated with larvae exposed to less than the 1000 ppm solution of chemical; larvae from the 1000 ppm solution were unable to cause gall formation on tomato roots.

## DISCUSSION

Cynem appears to be a promising nematicide since it can be easily applied to the soil, has low phytotoxicity and has long residual nematicidal activity. However, its mammalian toxicity is high, comparable to parathion, so its use involves hazard.

The halogenated hydrocarbons in use as nematicides today are fairly volatile and depend on this property for diffusion from the point of application for thorough soil treatment. The organic phosphates, being non-volatile, depend upon the thoroughness with which they are mixed with the soil for efficiency in killing nematodes. Also, the nematodes are present in the soil moisture and it is in this medium that the nematicide must contact the nematode, so some degree of water solubility is necessary.

Besides being a good nematicide, Cynem seems to have systemic insecticidal activity in tobacco. In the greenhouse, control of aphids on tobacco was excellent for the 38 days that the test lasted, but it remains to be seen if this activity occurs under field conditions and will continue until the plants reach maturity. Aphids cause the most severe damage to tobacco in the field near harvest.

If Cynem does remain systemic in the plant until harvest time, residues toxic to humans might persist in the cured leaf. No technique was available at this time for tracing the chem-

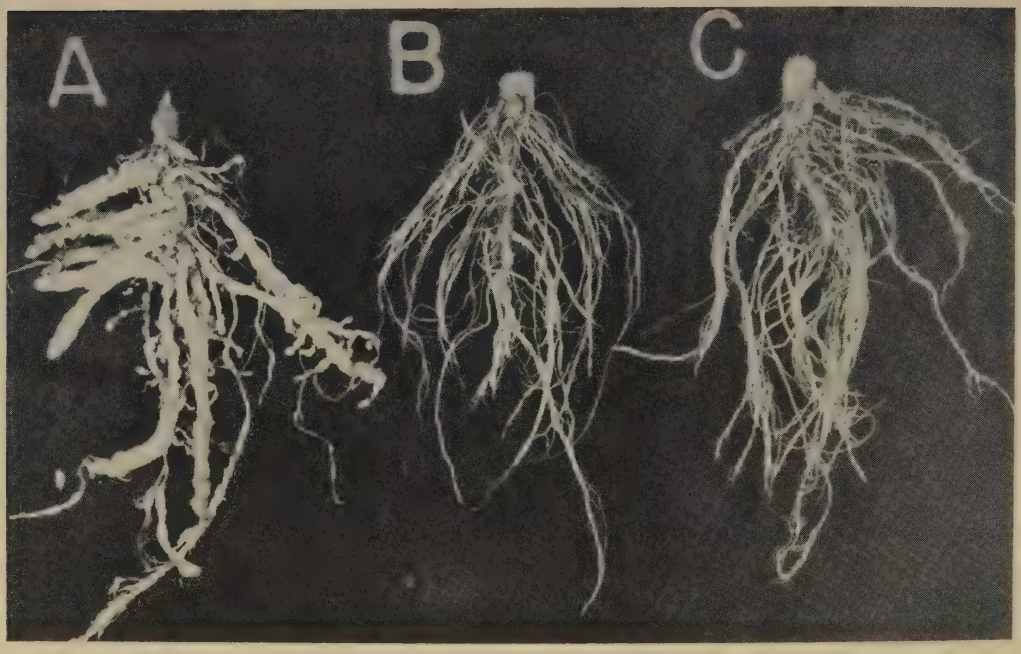


FIGURE 1. Roots of tomato plants from soil inoculated with root-knot nematodes 12 weeks after the soil was treated with Cynem. A -- check, B -- 10 pounds/acre, C -- 20 pounds/acre.

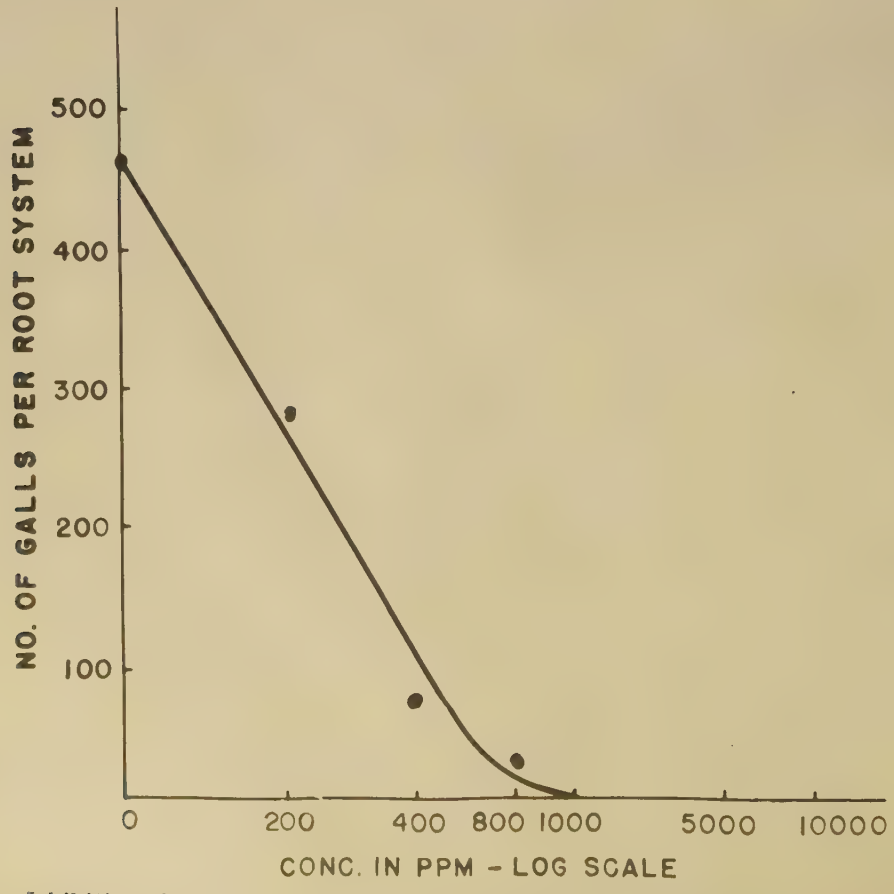


FIGURE 2. Inhibition of gall development on tomato roots inoculated with egg masses of root-knot nematodes treated with several concentrations of Cynem in water solution.

Table 1. Effects of concentration in solutions of Cynem on the motility of root-knot nematode larvae and their ability to cause gall formation on tomato roots.

| Cynem (ppm) | Time required for inactivation (hours) | Mean R-K index |
|---|---|---|
| 0 | -- | 2.9 |
| 1 | 4 | 3.0 |
| 10 | 2 | 3.0 |
| 100 | 1 | 2.7 |
| 1000 | 1 | 1.0 |

1% level of significance, using Duncan's range test
C.V. = 10%

ical and its by-products in the plant during the growing season.

The long residual nematicidal activity of Cynem and its low phytotoxicity are desirable characteristics for a chemical to be used for treating soil before establishing new plantings of orchards, strawberries, nursery stock and other perennials where nematode control is required for a period of time long enough to allow the plants to become established and vigorous. The use of Cynem for control of all types of nematode problems needs further investigation. Since this chemical has such long residual nematicidal activity and low phytotoxicity it might be used as a drench treatment around shrubs or trees, or sidedressed around annual or perennial crops. Similar compounds have proved effective for treating soil infested with nematode species that reproduced rapidly to large numbers following fumigation with halogenated hydrocarbons (4).

McBeth and Bergeson (8) in their nematicide screening program discard all chemicals with a lower basal toxicity than twice that of 1,3-dichloropropene-1,2-dichloropropane (D-D), which is lethal to D. dipsaci in 24 hours at 400 ppm in aqueous solution. Some chemicals might affect nematode feeding, reproduction or penetrability without affecting motility. Materials might also be nematistatic until fairly high concentrations are used and then nematicidal. Cynem in solution seems to have a nematistatic action on root-knot larvae until a concentration of 1000 ppm or more is reached, when it becomes nematicidal as indicated by the in vitro studies. Larvae exposed to concentrations of this chemical lower than 1000 ppm were inactivated after 1 to 4 hours depending on concentration, but when washed after 24 hours' immersion in chemical they were still able to penetrate and cause galling of tomato seedling roots. This indicates that care should be exercised when basing nematicidal activity of a chemical on inhibition of nematode motility.

In the soil, Cynem might be only nematistatic at the concentrations used. If the pounds per acre of soil is computed to a depth of 6.6 inches, which is about as deep as the chemical is thought to be mixed into the soil by disking, the amount of Cynem distributed in the soil at the 10 pound per acre rate would be around 5 ppm and at the 20 pound per acre rate 10 ppm. The residual activity of this chemical apparently prevents root-knot nematodes from entering plants. It might be interesting to see how long root-knot larvae would be inhibited in the soil at these various concentrations of chemical before death occurred.

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# GREEN TOMATO FRUITS -- A MEDIUM FOR INDUCING FRUIT ROT AND ASEXUAL SPORULATION WITH FUNGI ISOLATED FROM CLOVERS[1]

R. A. Kilpatrick[2]

## Summary

Isolates of Cladosporium sp., Fusarium oxysporum, F. roseum, F. solani, and Gliocladium roseum from Ladino white and red clovers induced severe rotting in green tomato fruits inoculated by toothpick and hypodermic needle methods. Rotting to a lesser extent was obtained with Curvularia trifolii and Trichoderma sp. Macroconidia of Fusarium spp. developed abundantly on inoculated fruits, but none of the other fungi sporulated or produced the perfect stage.

## INTRODUCTION

Green tomato fruits have been used to obtain the perfect stage of Gibberella. Boothroyd (1) obtained macroconidia and perithecia of G. zeae (Schw.) Petch within 3 weeks after inoculating green tomato fruits. Infection induced a dark-brown rot surrounding inoculated tissue.

This study was made to determine whether fungi isolated from Ladino white clover (Trifolium repens) and red clover (T. pratense) would produce: 1) the perfect stage on green tomato fruit and 2) rotting of inoculated fruit.

## MATERIALS AND METHODS

Two varieties of tomatoes, Eastern States Hybrid and Marglobe, were grown in 8-inch pots of soil in the greenhouse. When tomato fruits were approximately 1 inch in diameter they were inoculated with different fungi isolated from clovers.

All fungi were obtained from leaves or roots of Ladino white and red clover. These consisted of Cladosporium sp., Curvularia trifolii (Kauff.) Boed., Fusarium oxysporum Schlecht., F. roseum Lk., F. solani (Mart.) Appel & Wr., Gliocladium roseum (Lk.) Thom., Leptodiscus terrestris Gerdemann, Trichoderma sp., and Uromyces trifolii (Hedw. f.) Lév. var. fallens (Desm.) Arth.

A potato-dextrose broth (200 g potatoes, 10 g dextrose, and 1000 cc water) was dispensed into 250-ml flasks containing 6 or 7 pieces of toothpicks (1/2-3/4 inch long), which were then sterilized. After the flasks were cooled, spores and mycelium of the desired fungi were transferred to the broth and incubated at room temperatures for 5 to 7 days. Spores of the rust fungus were scraped from leaf lesions and transferred to water. A suspension of the spores was then injected into the fruits with a hypodermic needle. Individual, infested toothpicks were inserted into separate fruit on the plants, tagged, and examined daily. Control fruits were inoculated with sterilized toothpicks. Toothpicks were later omitted and a hypodermic needle was used to inject a spore and mycelial suspension into the fruit. Control fruits were injected with sterile distilled water. Following inoculation, fruits remained on the plants in a greenhouse (minimum of 65° F). Results in all experiments were obtained not later than 14 to 20 days after inoculation.

## EXPERIMENTAL RESULTS

With the exception of Fusarium spp. no sporulation of fungi occurred on inoculated fruits. Abundant macroconidia of Fusarium spp. developed on infected tissue. None of the fungi produced the perfect stage, but they caused various symptoms.

Five of the nine fungi tested (Table 1), Cladosporium sp., Fusarium oxysporum, F. solani, F. roseum, and to a lesser extent Gliocladium roseum, produced severe rotting within 20 days. The fungi were reisolated from infected tissue. Isolates of F. oxysporum (Fig. 1), F. roseum, and G. roseum caused a watery soft rot. This symptom was visible 7 days after inoculation. F. solani isolates produced a dry rot (Fig. 2). Infected fruit dried up and formed a mummy.

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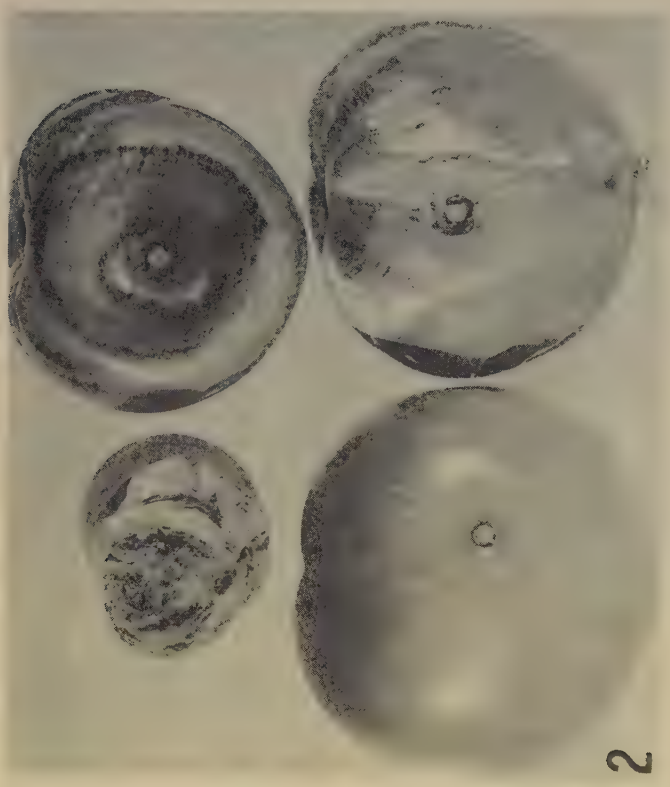
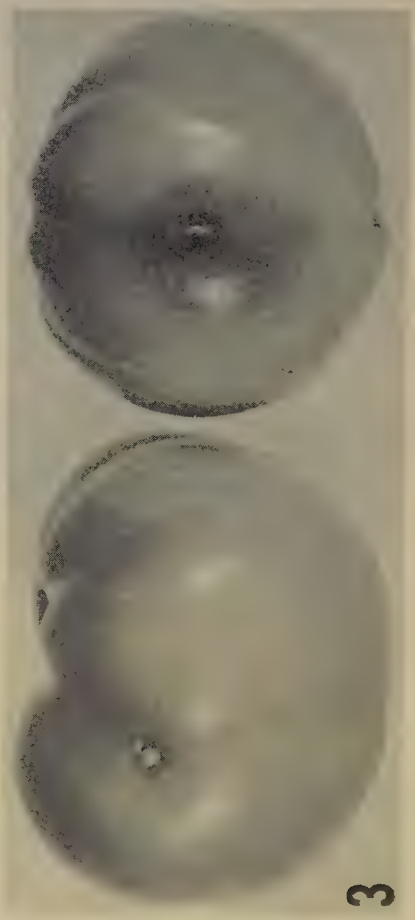
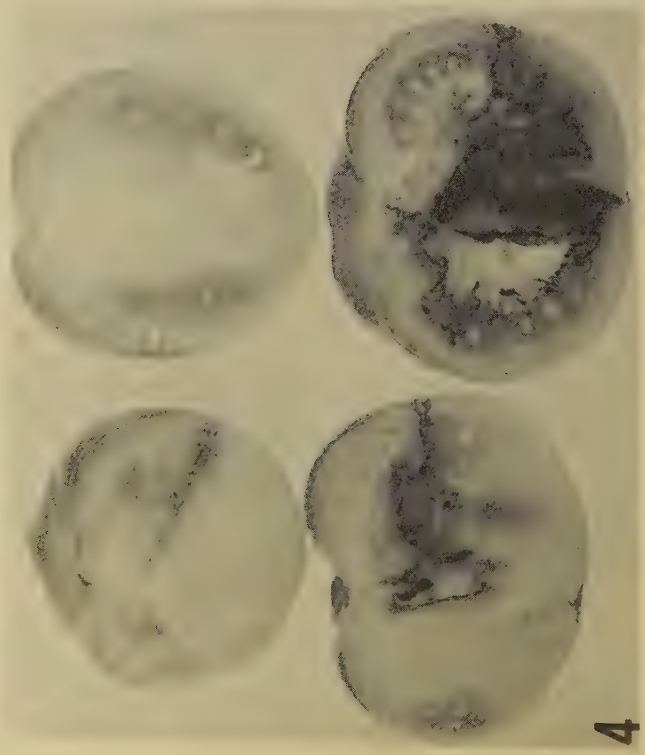
[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and Departments of Botany and Agronomy, New Hampshire Agricultural Experiment Station, Durham. Published with the approval of the Director of the New Hampshire Agricultural Experiment Station as Scientific Contribution No. 260.

[2]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.







(See legends on opposite page)

Table 1. Amount of tomato fruit rot produced by different fungi isolated from clovers.

| Fungus | No. of fruit inoculated | Severity of fruit rot[a] |
|---|---|---|
| None | 12 | 1.0 |
| Curvularia trifolii | 2 | 2.0 |
| Cladosporium sp. | 14 | 4.0 |
| Fusarium oxysporum | 19 | 5.0 |
| F. roseum | 4 | 4.5 |
| F. solani | 9 | 5.0 |
| Gliocladium roseum | 2 | 3.0 |
| Leptodiscus terrestris | 2 | 1.0 |
| Trichoderma sp. | 5 | 2.0 |
| Uromyces trifolii var. fallens | 5 | 1.0 |

[a]Based on a rating of 1 (healthy) to 5 (severe rotting).

Fruits inoculated with Cladosporium sp. showed a black discoloration of the external area surrounding the toothpick (Fig. 3). However, the internal area was severely rotted (Fig. 4). The internal symptom was typical of that described by Gardner (2) for Cladosporium fulvum Cke. When tomato plants were inoculated with Cladosporium sp., leaf symptoms (typical of C. fulvum) developed only after 5 days' incubation in a moist chamber. Symptoms failed to develop when plants were incubated for only 3 days. Identified isolates of C. fulvum and the isolate from clovers were not compared. Curvularia trifolii and Trichoderma sp. produced a black discoloration of the internal area surrounding the toothpick wound. No rotting or discoloration was noted in fruits inoculated with sterile water, Leptodiscus terrestris, or Uromyces trifolii var. fallens.

## DISCUSSION

The amounts of rot induced on green tomatoes by fungi from clovers do not indicate their relative pathogenicity on this host. They do show, however, that injured tomato fruits provide a favorable medium for some fungi. This, in turn, results in deterioration of host tissue.

The species of Cladosporium used in this study was not identified. However, the fruit and leaf symptoms and requirement of a prolonged high humidity for initial leaf infection suggests a close relationship of species isolated from clovers to C. fulvum. Cladosporium sp. has been isolated from all parts of clover plants (3), but disease symptoms have not been described on this host.

The two methods of inoculation offer a rapid evaluation of fungus specificity on tomato fruits. The toothpick method would be most reliable, since drying of host tissue would be minimized.

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FIGURE 1. Soft rot on green tomato caused by Fusarium oxysporum (right); uninoculated (control) left.

FIGURE 2. Mummification of green tomato caused by Fusarium solani; uninoculated (control) lower left.

FIGURE 3. Black discoloration on green tomato caused by Cladosporium sp., (right); uninoculated (control) left.

FIGURE 4. Stages of internal dry rot of green tomato caused by Cladosporium sp.; uninoculated (control) upper right.

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# CONTROL OF BACTERIAL SCAB AND FUSARIUM CORM ROT OF GLADIOLUS

Lester P. Nichols[1]

Bacterial scab, caused by Pseudomonas marginata (McCull.) Stapp, is one of the most common diseases of gladiolus in Pennsylvania. The neck rot phase of the disease may cause serious reductions in flower production; the lesions on the corms, while usually not affecting spike yield, may increase the susceptibility of corms to storage rot organisms and spoil the appearance of the corms for sale.

Corm rot and yellows, caused by Fusarium oxysporum f. gladioli (Massey) Snyd. & Hans., results in severe losses in spike and corm production.

In 1959 a non-replicated trial for the control of bacterial scab was conducted in a commercial gladiolus field in Somerset County, Pennsylvania[2]. Corms treated with Delsan A-D, Emmi, and those treated with Emmi and planted in soil treated with heptachlor, were compared with the "standard" corm treatments of calomel and New Improved Ceresan, and an untreated check. The results of this test showed that the Emmi corm treatment planted in heptachlor treated soil had a delaying effect on the emergence of the plants. There was no outstanding difference in total spike production between any of the treatments although a delay of flowering of up to 2 weeks with all the treatments containing mercury was observed. Corms treated with Delsan A-D produced corms that had 36% scab, while those produced by corms treated with calomel had 79%. Due to the low incidence of Fusarium corm rot and yellows, no information on the control of this disease was obtained.

In 1960 the test was repeated. The preplanting treatments were as follows:

1. - Delsan A-D (60% thiram[3], 15% dieldrin[3]) - 2 level tablespoons per 100 corms, dusted on the corms by shaking in a bag.
2. - Panoram D-31 (56.2% thiram, 18.8% dieldrin) - 2 level tablespoons per 100 corms, dusted on in a bag.
3. - Emmi (10.34% N-ethylmercuri-1,2,3,6-tetrahydro-3,6-endomethano-3,4,5,6,7,7-hexachlorophthalimide) - 1 cup per 25 gallons of water, 2-hour soak of corms.
4. - Emmi, corm soak as in treatment number 3; corms planted in furrows treated with 5% granular heptachlor (3a,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene), 63.6 grams sprinkled per 100 feet of row.
5. - Phaltan 75% (N-trichloromethyl thiophthalimide) - 2.5 pounds per 25 gallons of water plus Ortho Spray Sticker, 3 cups per 25 gallons, 30 minute soak of corms.
6. - New Improved Ceresan (5% ethyl mercury phosphate) - 1/2 pound per 25 gallons of water, 15-minute soak of corms.
7. - Ceresan M(7.7% ethyl mercury p-toluene sulfonanilide) - 1/2 pound per 25 gallons of water, 15-minute soak of corms.
8. - Untreated check.

The corms were planted May 19, 1960 in non-replicated rows. Each treatment consisted of 100 corms each of the varieties Gold and White Excelsior planted in a 100-foot row. Treatments were compared in adjacent rows for one variety; the other variety was compared in the next 100 feet of the same rows but the order of the treatments was reversed.

## RESULTS

Effects on Emergence: Emergence counts made on July 15 are summarized in Table 1. The relatively poor stand of plants of the variety Gold in all treatments apparently was due to the high incidence of Fusarium oxysporum f. gladioli carried within the corms. Although these corms did not show visible symptoms of Fusarium corm rot at planting, only 62% of the untreated corms produced plants and all of these either died or were rogued because they showed symptoms of Fusarium yellows.

Effects on Spike Production: The number of saleable spikes produced by each treatment is shown in Table 1. In the White Excelsior variety the number of spikes produced from corms treated with Panoram D-31, and Delsan A-D were outstanding when compared with the other treatments. Even in the variety Gold where the plant count was reduced by Fusarium, the

[1]Associate Professor, Plant Pathology Extension, The Pennsylvania State University, University Park, Pennsylvania.

[2]Nichols, Lester P. 1960. Corm and soil treatment for the control of bacterial scab of gladiolus. Plant Disease Reptr. 44: 417-418.

[3]thiram = tetramethylthiuram disulfide; dieldrin = 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo-exo-5,8-dimethanonaphthalene.

corms treated with Panoram D-31 and Delsan A-D gave better spike production than the other treatments.

Disease Control: The corms were harvested on October 7. On December 14 all of the new corms of each variety were husked and checked for the presence of scab and Fusarium corm rot.

Scab: The corms were grouped into classes from 0 to 5, depending on the severity of scab lesions. The classes used as a basis for a mean disease rating were as follows: 0, corms free of scab lesions; 1, corms showing not more than 2 scab lesions not larger than 1/8 inch in diameter; 2, corms showing not more than 3 to 8 scab lesions up to 1/4 inch in diameter; 3, corms showing scab lesions on not more than 1/3 of their surface; 4, corms showing scab lesions on not more than 2/3 of their surface; 5, corms severely scabbed. To determine the severity of scab for each treatment a mean disease rating, shown in Table 1, was calculated by multiplying the number of corms in each class by the class number, summing the products, and dividing by the number of corms checked. A mean disease rating of 0.0 would indicate that all the corms were free of scab lesions, and a mean disease rating of 5.0 that all the corms checked were severely scabbed. With both varieties, the Delsan A-D and Panoram D-31 treatments gave much better control of scab than did any of the other treatments.

Fusarium Corm Rot: Data on the incidence of Fusarium corm rot are shown in Table 1. In the variety Gold all of the treatments containing mercury gave good control; Delsan A-D, Panoram D-31, and Phaltan gave poor control. In the variety White Excelsior all of the treatments containing mercury, with the exception of Emmi-heptachlor, gave better control than did Delsan A-D and Panoram D-31.

Table 1. Effect of gladiolus corm treatment on stand, spike production, control of scab and corm rot, 1960.

| Treatment | Results - based on 100 corms planted per treatment: Stand | Saleable spikes | Total new corms | % rot-free corms | % scab affected corms | Scab rating | % Fusarium rotted corms |
|---|---|---|---|---|---|---|---|
| | | | Variety Gold | | | | |
| Delsan A-D | 59 | 27 | 47 | 39 | 35 | 0.4 | 35 |
| Panoram D-31 | 76 | 29 | 75 | 20 | 52 | 0.5 | 52 |
| Emmi | 57 | 9 | 46 | 0 | 100 | 2.3 | 0 |
| Emmi + heptachlor | 31 | 3 | 26 | 0 | 96 | 2.2 | 8 |
| Phaltan | 75 | 21 | 62 | 2 | 85 | 2.0 | 26 |
| New Improved Ceresan | 57 | 10 | 54 | 6 | 85 | 1.6 | 2 |
| Ceresan M | 65 | 17 | 45 | 7 | 93 | 1.8 | 0 |
| Check | 62[a] | - | - | - | - | - | - |
| | | | Variety White Excelsior | | | | |
| Delsan A-D | 100 | 74 | 97 | 7 | 90 | 1.9 | 6 |
| Panoram D-31 | 99 | 82 | 93 | 11 | 85 | 1.6 | 7 |
| Emmi | 91 | 25 | 85 | 0 | 100 | 3.6 | 0 |
| Emmi + heptachlor | 49 | 14 | 45 | 7 | 87 | 2.5 | 11 |
| Phaltan | 90 | 42 | 76 | 0 | 96 | 4.2 | 0 |
| New Improved Ceresan | 95 | 51 | 97 | 0 | 100 | 3.8 | 0 |
| Ceresan M | 89 | 53 | 84 | 0 | 100 | 5.0 | 0 |
| Check | 64 | 42 | 54 | 0 | 94 | 4.1 | 11 |

[a]Plants all killed by Fusarium before harvest time.

## DISCUSSION

In 1959 and 1960 a preplanting dust treatment of thiram-dieldrin gave excellent control of bacterial scab and an outstanding increase in spike production as compared with the other treatments used. In 1960 the incidence of Fusarium corm rot in one variety was high and the control of this disease by the thiram-dieldrin treatments was poor. The treatments containing mercury gave excellent control of the Fusarium corm rot. In 1959 and 1960 the treatments containing mercury delayed the date of flowering up to 2 weeks as compared with other treatments and the total spike production was poor. Since both bacterial scab and Fusarium corm rot are problems in the commercial gladiolus fields and home gardens of Pennsylvania the ultimate in a preplanting treatment will be one that gives excellent control of both these diseases and at the same time results in high spike yields and gives no delay in the date of flowering.

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## PHYTOTOXICITY OF PHENYLMERCURY SPRAYS TO WHITE OAK FOLIAGE

Lester P. Nichols[1]

Phenylmercury sprays are recommended as fungicidal sprays for the control of white oak anthracnose, caused by Gnomonia veneta (Sacc. & Speg.) Kleb. (1, 2, 3, 4, 5). Timing recommendations range from one spray applied at bud break to three to four sprays applied at the dormant stage through the period of new leaf development.

In late May of 1959 leaves showing marginal necrosis and downward curling, with defoliation in advanced stages, were observed in an estate planting of large white oak trees in western Pennsylvania. The trees had been sprayed at bud break, 14 days later, and about 20 days later with 7.5% phenyl mercuri triethanol ammonium lactate (Puratized Agricultural Spray) at the rate of 1 pint/100 gallons of spray. The injury was not present on all the trees and, in some cases, branches of trees showing foliage injury were interlaced with branches of adjoining trees which apparently were unaffected. The foliage of several hickory trees within the white oak planting showed a small amount of similar injury. Also, an unsprayed red maple at the edge of the white oak planting showed marginal necrosis of the leaves. A laboratory analysis of foliage and twigs taken from the maple showed that there was no mercury present in the samples. In 1960 the trees were not sprayed and developed normal foliage.

## MATERIALS AND METHODS

In the spring of 1960 a test was arranged to determine if Puratized Agricultural Spray at the recommended rate of 1 pint/100 gallons of spray, or at higher concentrations, might cause injury to white oak foliage. White oaks, 6 to 10 feet tall, in a woodlot of the Pennsylvania State University were sprayed four times with Puratized Agricultural Spray at 1, 2, 4, 8 and 16 pints/100 gallons of spray. The fungicide at each concentration was applied to four separate branches of the same or adjacent trees. Applications were made with a trombone-type manual sprayer, spraying to the point of run-off. At the time of each application, four branches of one tree were sprayed with water to serve as a control. Branches of nearby trees were marked to serve as unsprayed controls.

Applications were made as follows: April 23, as most of the buds were swelling, air temperature 80° F; April 30, some trees with buds just opening, others with leaves up to 0.75 inches long, temperature 65°; May 20, on leaves ranging from 1 inch to one-third grown, temperature 75° and leaves wet from recent shower; June 4, on fully grown leaves, temperature 75°.

## RESULTS AND CONCLUSIONS

Observations of the sprayed trees on April 30, 1 week after the first application, did not show a phytotoxic reaction for any of the concentrations used.

On May 20 a slight yellowing and downward curling of the leaves was observed on

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branches sprayed twice at the 16 pints/100 gallon concentration.

Observations on June 4, after three applications were made, showed the following: 1 pint - slight marginal necrosis of five to six leaves on two of the four branches; 2 pints - downward curling and marginal necrosis of half of the leaves on one branch, small brown blotches in the center of three leaves on one branch; 4 pints - downward curling and marginal necrosis of from 2 to 50% of the leaves on the four branches; 8 pints - downward curling with marginal necrosis and stunting of all leaves on three branches, 95% of leaves dead on one branch; 16 pints - downward curling, marginal necrosis and stunting of from 85 to 95% of the leaves on three branches, 10% of leaves curled and brown with the remainder stunted on one branch.

On June 11, after four applications were made, the following injury was noted: 1 pint - small round yellow blotches on 10% of the leaves on all four branches; 2 pints - small round blotches on 15% of the leaves on all four branches; 4 pints - downward curling, marginal necrosis and stunting of all the leaves on all four branches; 8 pints - downward curling, marginal necrosis and stunting of all leaves on three branches, 99% of leaves dead on one branch; 16 pints - severe curling, marginal necrosis and stunting of all leaves on all four branches.

No injury was observed at any time on the control branches sprayed with water. Anthracnose did not appear on any of the sprayed or unsprayed trees in the study area although several large trees less than a mile distant were severely defoliated by anthracnose by June 11.

Under the conditions of this test Puratized Agricultural Spray at 1 pint/100 gallons of spray was not phytotoxic during the first two applications and developed only very slight injury following the third and fourth applications. A wide range of safety was indicated.

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# PERCENTAGE YIELD LOSS AS RELATED TO PERCENTAGE LOOSE SMUT IN BARLEY

Donald J. Morton[1]

## Summary

Portions of 10 seed samples with different smut percentages were disinfected by an anaerobic treatment and compared with non-treated portions in yield tests. A regression coefficient for the average yield losses and smut percentages shows the percentage loss increased 0.86 with each 1% increase in smut incidence. This figure approximates those found in earlier experiments using other methods, and suggests that each percent of barley loose smut generally causes slightly less than 1% yield reduction.

## INTRODUCTION

The relationship between smut incidence and yield loss has been studied in several cereal crops. Working with wheat bunt, Slinkard and Elliott (10) mixed seed of a susceptible variety in varying proportions with a resistant back-cross derivative and calculated a loss of 0.77% for each percent increase in smut. Other workers who studied this disease included Heald and Gaines (4) who found a value of 0.89%, Flor, et al. (3) who showed regression coefficients of 1.26 for a susceptible and 0.76 for a moderately susceptible variety, and Leukel (5) who reported the average percentage of bunt was slightly greater than the percentage reduction in yield. Wood (11) investigated loose smut of oats by applying various chemical seed treatments, and obtained losses of 1.00, 0.76 and 1.02% for each percent smut at levels of 2, 33, and 41% smut, respectively.

Brown (1) estimated losses caused by loose smut of winter wheat by cutting off different percentages of heads at heading time. Results indicated that the percentage reduction in yield was approximately the same as the percentage reduction in heads, and that the closeness of the approximation was influenced by seasonal and varietal differences. Compton and Caldwell (2) also worked with loose smut, and compared yields of winter wheat having different levels of natural infection. They concluded that the percentage loss was about the same as the percentage smut in the field.

Few studies have been made on the effect on yield of barley loose smut, a disease caused by Ustilago nuda (Jens.) Rostr. Semeniuk and Ross (9) found a direct linear relationship between the percentage incidence of loose smut and the yield reduction at three locations; for each percent increase in smut the yield decreased 0.85% at St. Paul, Minnesota, 1.2% at Fallis, Alberta, and 1.4% at Edmonton, Alberta. They mixed different ratios of smut-free and artificially-inoculated seed of comparable size and genotype to obtain a range of infection levels, a procedure which reduced natural variability between the smut-free and smutted seed.

A method using seed disinfection for estimating the effect of loose smut on barley production was tried at Fargo in 1960. This procedure removed the possibility that infected and non-infected seed were initially different in any way, but introduced the variable of the treatment. It was conjectured that results obtained with this method could be compared with previous information found by other means.

## MATERIALS AND METHODS

Seed samples representing 10 different farms and shown by embryo examinations (6, 8) to have different levels of U. nuda infection were divided into two portions, one of which was disinfected by an anaerobic method (7) while the other was left untreated. A control lot of seed with no infection was handled similarly to determine whether the treatment alone might influence yield.

Each seed sample was planted individually in a randomized block design in which the treated and untreated portions were each replicated eight times. A replication comprised the center eight feet in each of two adjacent 10-foot rows, making a total of 16 feet of row per replication. Rows were planted 1 foot apart, and a guard row of winter wheat was grown between replications.

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Research supported by the Malting Barley Improvement Association.

Smut percentages were estimated by the proportion of smutted heads in each row, and ranged from 4.2 to 27.8% in the untreated plots. No smut was found in any of the treated rows. All seed were of the variety Traill, except for those with 7.7 and 11.7% smut where Forrest and Kindred, respectively, were represented.

Yield was regarded as the weight of grain per replication, and the difference in yield between the smutted and smut-free plots was divided by the yield of the treated plots, multiplied by 100 and considered as percentage loss. The effect of the disease on kernel plumpness was evaluated by screening the grain into plump, medium and thin fractions for weighing.

## RESULTS

The results of the experiment are presented in Table 1, where the average pounds of barley produced by the treated and untreated portions of each seed lot are listed, and where the percentage loss is compared with the percentage smut. In general, percentage loss in the smutted plots was a little less than the percentage of smut, although in two cases (4.2 and 12.0% smut) it was greater.

Data for each seed lot were analyzed independently and, because of field variations, significance at the 5% level was not obtained with the four lowest infection levels (4.2, 5.2, 7.7 and 9.1% smut). However, the relationship between yield loss and smut incidence was generally constant and the regression coefficient for the means of percentage loss and percentage smut for each seed lot shows a percentage loss increase of 0.86 for each percent increase of the disease.

The treated Check plots produced 2.6% more grain than the untreated, a difference not significant at the 5% level.

No consistent differences were found in kernel size between treated and untreated plots.

Table 1. Yields from treated and untreated plots with different loose smut percentages.

| Seed lot no. | Variety | Average yield/16-foot row[a] (in pounds) | | | % loss | %[b] smut in field | % loss for each % smut |
|---|---|---|---|---|---|---|---|
| | | treated | untreated | loss | | | |
| 1 | Traill | 385 | 367 | 18 | 4.7 | 4.2 | 1.12 |
| 2 | Traill | 417 | 399 | 18 | 4.3 | 5.2 | 0.83 |
| 3 | Forrest | 337 | 318 | 19 | 5.6 | 7.7 | 0.73 |
| 4 | Traill | 297 | 280 | 17 | 5.7 | 9.1 | 0.63 |
| 5 | Kindred | 412 | 374 | 38 | 9.2* | 11.7 | 0.79 |
| 6 | Traill | 305 | 261 | 44 | 14.4** | 12.0 | 1.20 |
| 7 | Traill | 450 | 391 | 59 | 13.1** | 14.6 | 0.90 |
| 8 | Traill | 432 | 363 | 69 | 16.0** | 20.9 | 0.77 |
| 9 | Traill | 437 | 353 | 84 | 19.2** | 21.4 | 0.90 |
| 10 | Traill | 304 | 232 | 72 | 23.7* | 27.8 | 0.85 |
| Average | -- | -- | -- | -- | 11.6 | 13.5 | 0.86 |
| Check | Traill | 379 | 369 | 10 | 2.6 | 0.0 | -- |

[a]Average of eight replications.
[b]No smut appeared in any of the treated plots.
* "F" value significant at 5% level.
** "F" value significant at 1% level.

## DISCUSSION

Seed that initially were identical in every respect were compared by disinfecting part of each seed sample and comparing it with the remaining smutted seed. The disinfection, of course, introduced a source of variation but on the basis of the check seed lot it is unlikely that yield was appreciably altered.

The regression coefficient for the average yield loss and smut percentage shows that the percentage loss increased 0.86 with each increase of 1% in smut incidence. This figure is very close to the coefficient of 0.85 found for barley loose smut by Semeniuk and Ross (9) at St. Paul, and generally approximates the yield losses caused by wheat bunt, oat loose smut

and wheat loose smut mentioned in the Introduction. These similar findings using a different procedure support the belief that under most conditions tested each percent smut causes a little less than 1% yield loss.

The wide range between the regression coefficients for the different seed lots suggests that the seed source might influence the yield loss-smut relationship. Also, the relationship is apparently more consistent in the seed lots with higher smut percentages where results were most significant statistically.

The lack of a consistent difference in kernel plumpness between treated and untreated plots does not support the premise that heads adjacent to smutted ones receive more nutrients and grow larger, with a resulting reduction in yield loss. However, only small differences in this characteristic would be needed to cause such an effect on yield, and field variations may have prevented recognition of such a trend.

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# ADDITIONAL JUNIPER HOSTS OF CEDAR-APPLE AND CEDAR-HAWTHORN RUSTS

Dan Neely and E. B. Himelick[1]

Abstract

Observations in 1960 of the Juniperus collection at the Morton Arboretum near Lisle, Illinois showed that four varieties of Juniperus species not previously so reported are susceptible to cedar-apple rust. Two species and 11 varieties of other Juniperus species not reported in other host lists are susceptible to cedar-hawthorn rust.

An extensive collection of native and naturalized species, varieties, and forms of junipers are growing in the Morton Arboretum near Lisle, Illinois. Observations on the incidence of cedar-apple rust, caused by Gymnosporangium juniperi-virginianae Schw., and cedar-hawthorn rust, caused by G. globosum Farl., on individual plants in this collection were recorded on May 22, 1959. The results of these observations and the reports of susceptibility and resistance of junipers by other observers were published in 1960 (2). Again on April 29, 1960, the plants in the juniper collection at the Morton Arboretum were examined. This examination was made immediately following a warm spring rain. A few junipers assumed to be resistant in 1959 were found to be susceptible in 1960.

Table 1. Junipers resistant (R) or susceptible (S) to cedar-apple and cedar-hawthorn rusts.

| Species, varieties, and forms of Juniperus | Cedar-apple rust | Cedar-hawthorn rust | Species, varieties, and forms of Juniperus | Cedar-apple rust | Cedar-hawthorn rust |
|---|---|---|---|---|---|
| J. chinensis L. | | | J. scopulorum Sarg. | | |
| f. globosa (Hornibr.) Rehd. | | S - 1[a] | hilli D. Hill | | S - 1 |
| f. mas (Gord.) Rehd. | R | R | hilli argentea pyramidalis | | S - 1 |
| f. pendula (Franch.) Beiss. | R | R | moffeti Hort. | | S - 1 |
| Pfitzeriana nana | R | R | pendula D. Hill | | S - 1 |
| var. plumosa aurea Hornibr. | R | R | J. squamata Lamb | | |
| var. Sargenti Henry | S - 1 | S - 1 | var. Fargesii Rehd. & Wils. | R | R |
| J. communis L. | R | R | J. virginiana L. | | |
| f. aureo-spica (Rehd.) Lipa | R | R | (Berg's strain) | R | S - 1 |
| f. oblongo-pendula Beiss. | R | R | f. albo-spica Beiss. | S - 2 | S - 1 |
| J. horizontalis Moench | | | cupressifolia | S - 1 | S - 1 |
| f. alpina (Loud.) Rehd. | | S - 1 | nova D. Hill | | S - 2 |
| f. plumosa Rehd. | S - 1 | | f. reptans (Beiss.) Rehd. | | S - 3 |
| J. pinchotii Sudw. | | S - 1 | f. Schottii (Gord.) Beiss. | | S - 1 |

[a]S - 1, slight infection; S - 2, moderate; S - 3, severe. Plants that showed the same resistance or susceptibility in 1960 as in 1959 (2) are not included in this report.

The disease reactions of the species, varieties, and forms of junipers listed in Table 1 are additions or changes to the more extensive listing which was published in 1960 (2). Four varieties or forms of Juniperus species are reported for the first time as susceptible to cedar-apple rust. Juniperus pinchotii, one variety and one form of J. chinensis, and 11 varieties or forms of other Juniperus species are susceptible to cedar-hawthorn rust. Eight varieties or forms of junipers, seven of which were included in the previous listing, were examined for the first time in 1960 in the Morton Arboretum. They appear to be resistant to both rust diseases. Berg's selection of Juniperus virginiana (1) is resistant to cedar-apple rust and susceptible to cedar-hawthorn rust.

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# OBSERVATIONS ON FOMES ANNOSUS ROOT-ROT IN NATURAL STANDS OF LOBLOLLY AND SHORTLEAF PINE

Chas. H. Driver and T. R. Dell

The root-rot disease caused by the fungus Fomes annosus (Fr.)Cke. has induced mortality in natural mixed stands of loblolly (Pinus taeda) and shortleaf pine (Pinus echinata) within the Southlands Experiment Forest in Decatur County, Georgia.

The stations at which these observations were made exhibit the following points of interest:

1. A natural mixed stand of the 40-year age class contained two groups of dead trees (Figs. 1 and 2). Cutting, as indicated by old stumps in the immediate vicinity of the groups of dead trees, had been carried out several years ago. A woods' road construction operation in 1958 resulted in another light cutting, exposing stumps in the same area. The site of the stand where the mortality occurred was of a low-land, moist type with a light sandy surface soil over an imperfectly drained yellow friable sandy clay subsoil. The loblolly site index was determined to be 95 feet on a 50-year basis.

The larger of the two groups (Fig. 1) contained eight dead trees ranging in size from 4 to 15 inches, diameter breast high. The average height of the merchantable size trees was 85 feet. This mortality represents a volume loss of 1188 board feet or 2.9 cords within an area of approximately one-fourth acre.

Several pine stumps in the area exhibited numerous fruiting bodies of F. annosus (Fig. 3). The proximity of the stumps indicates that they probably functioned as the source of initial infection. The disease inducing fungus then spread to the living trees probably by natural root grafts. Living trees of both species in the area of infestation exhibited fruiting bodies of F. annosus at the root collars within the needle duff.

2. Figure 4 illustrates mortality of loblolly pine thought to be induced by annosus root-rot. Again, the location is within a limited area logged for woods' road construction. The mixed stand of loblolly and shortleaf pine has an average age of 30 years. Ten dead trees in this group and several stumps in the immediate vicinity showed fruiting bodies of F. annosus. The dead trees of the area had a range of diameters at breast height of 5 to 15 inches. The volume loss was 576 board feet, or 1.7 cords within an area of approximately one-fourth acre. The site of this stand is within a well drained ridge system with a loamy sand soil having a site index for loblolly pine of 100 feet, determined on a 50-year basis. Again, living trees within the area were observed bearing fruiting bodies of F. annosus.

3. Observations on a similar stand revealed that reproduction of both loblolly and shortleaf pine was being attacked by F. annosus (Fig. 5). Stumps and dead standing sawtimber size trees were observed bearing fruiting bodies of this fungus within the immediate vicinity of the infected pine saplings. The pine reproduction exhibiting symptoms of annosus root-rot varied in size from seedlings to saplings up to 10 feet in height and 3 inches in diameter at the base. The site on which this mortality occurred was a rather dry ridge top with a sandy loam soil expressing a site index for loblolly pine of 85 feet, determined on a 50-year basis. It is noteworthy that two species of the hardwood understory saplings at this location also showed fruiting bodies of F. annosus. The species infected were southern red oak (Quercus falcata) and shining sumac (Rhus copallina).

To the authors' knowledge this is the first of such observations to be reported for the State of Georgia.

The progressive development of the disease of annosus root-rot is being followed within the above-reported stands, as well as that previously reported[1] in slash pine plantations.

FIGURE 1. Mortality thought to be induced by annosus root-rot in sawtimber size trees of a natural mixed stand of loblolly and shortleaf pine.

FIGURE 2. Mortality of pine in a 40-year-old natural stand.

FIGURE 3. A pine stump within the area of Figure 1 exhibiting fruiting bodies of F. annosus.

FIGURE 4. Mortality within a 30-year-old natural mixed stand of loblolly and shortleaf pine. The white flags in the center foreground of the photograph mark the location of pine stumps exhibiting fruiting bodies of F. annosus.

FIGURE 5. A shortleaf pine sapling exhibiting a fruiting body of F. annosus at the ground line.

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[1]Driver, Chas. H., and T. R. Dell. 1961. Fomes annosus root-rot in slash pine plantations of the Eastern Gulf Coast States. Plant Disease Reptr. 45: 38-40.




(See legends on opposite page)

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# POWDERY MILDEW OF POTATO IN UTAH

Michael Treshow and Orson S. Cannon[1]

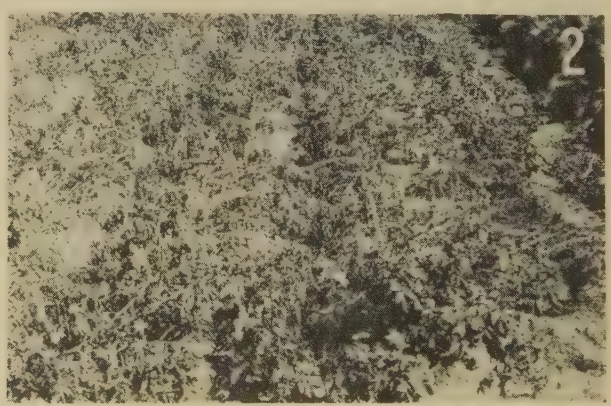
Wilting of potatoes, caused by a powdery mildew fungus, has caused concern among Utah growers for the past 7 or 8 years. Previously, the disease was reported in Kentucky (5), New Jersey (2), and Washington (3), but apparently no appreciable commercial damage was involved. In Palestine, however, Chorin (1) reported the Oidium stage to be widespread and destructive. In Utah damage from powdery mildew has annually become of increasing severity and prevalence. The past year all fields observed in Utah and Salt Lake Counties were infected.

## SYMPTOMATOLOGY

Powdery mildew symptoms generally first appear toward the middle of August or early in September, at which time the powdery white growth becomes most conspicuous along the petiole, midrib, larger veins, and stem (Fig. 1). Subsequently, both upper and lower leaf surfaces become covered with the white pubescence of the fungus mycelia. During this rather brief stage, the conidia are also prevalent. The whitish appearance soon gives way to chlorosis and necrosis as the leaf is killed. At the same time, infected areas of stem and petiole tissue are killed, leaving necrotic flecks and streaks which gradually coalesce to form larger lesions as the infection spreads. Within 2 to 4 weeks from the time of initial infection most, or all, of the vine is killed. Infection and subsequent vine collapse generally appear relatively uniformly over large areas of the planting or even over the entire planting (Fig. 2). The rapid dieback results in the collapse and destruction of entire plantings. By the time vines have collapsed, perithecia are prevalent over the dead leaves and stems.

FIGURE 1. Early stage of powdery mildew infection showing typical necrotic lesions produced along the petiole and midribs.

FIGURE 2. Late stage of injury where foliage is necrotic and vines collapsed. Parts of a few vines in the lower edge of the picture have escaped severe infection.



## CAUSAL ORGANISM

Perithecia range between 135 and 165$\mu$ in diameter, averaging 147$\mu$. They generally contain 5 to 10 asci ranging in size from 48 to 82$\mu$ by 26 to 43$\mu$ and averaging 36 by 64$\mu$. Asci contain oval to round ascospores ranging in size from 14 to 27$\mu$ by 14 to 19$\mu$ and averaging 16 by 22$\mu$. The fungus was identified as Erysiphe cichoracearum DC.

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## EPIDEMIOLOGY

Powdery mildew affects a number of different potato varieties, as well as other susceptible species, grown in Utah, including Kennebec, Pontiac, Red Bliss, and Idaho Russet. Menzies (3) reported the occurrence of mildew in Washington on the Mesaba, Warba, White Rose, and Russet Burbank varieties. Where adjacent fields of different varieties were inspected, Kennebec and Pontiac appeared to be the most susceptible varieties under Utah conditions. Red Bliss, which has now been largely replaced by Pontiac, was severely damaged in 1953. Russet appears to be relatively tolerant compared with other varieties grown in Utah.

The season in which infection occurs appears to be much the same each year and is apparently not greatly influenced by the variety or degree of plant maturity. Late planted potatoes, which are just setting tubers when the plants are hit, are most damaged. In such cases the vines completely collapse before the tubers have an adequate chance to size, and yields are markedly reduced. Where yields formerly ran around 20 tons per acre, some growers believe that mildew has cut production down to 10 tons. This may be true where plantings were set late in May, but where set 2 to 3 weeks earlier, such severe production losses are unlikely and other factors may be involved. Thirty percent yield reductions have been estimated in some Pontiac fields and may be even higher in some Kennebec plantings; furthermore, many of the tubers of severely affected plants may not attain a marketable size. Potatoes planted early in May have made much of their growth by the time mildew becomes severe, and yields in such plantings appear satisfactory. This suggests that early planting may provide some control. In Palestine, where powdery mildew was reported as destructive, Palti and Moeller (4) reported effective control with fine grade sulfur dusts. They suggested starting applications with the first appearance of symptoms and repeating at 10- to 14-day intervals until 3 to 4 weeks before harvest.

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# ON THE PROBLEM OF XYLOPOROSIS AND CACHEXIA DISEASES OF MANDARINS[1]

I. Reichert and A. Bental[2]

## Summary

A survey was made on the state of health of Clementine trees; 200 were grafted on sweet lime and 22 on sour orange rootstocks, all of the same age and growing under similar conditions.

The diseases affecting Clementine trees were: cachexia, xyloporosis and an undetermined disorder, characterized by inverse pitting.

Almost all sweet lime rootstocks exhibited symptoms of xyloporosis. In the scions, 20% of the trees showed xyloporosis and 47.5% cachexia. Inverse pitting, either alone or in association with cachexia or xyloporosis, was detected in 10% of the trees, suggesting that it might be a distinct disease.

The declining effect of cachexia was more marked than that of xyloporosis. Inverse pitting, alone or in association with other disorders, resulted in a most severe decline.

Clementine trees on sour orange rootstocks were 100% affected by cachexia in their scions, but the rate of decline was much lower than that on sweet lime: 55% versus 71% (47 + 24).

Xyloporosis in the scions was not responsible for the decline of the trees, as it did not additionally aggravate the decline already caused by the presence of xyloporosis in the rootstocks.

Our results seems to corroborate Grant's (3) contention that cachexia and xyloporosis are distinct, as both appear on Clementine trees of the same age, and growing under the same conditions.

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This paper may add some new facts to the clarification of the problem of xyloporosis and cachexia. Xyloporosis was discovered in Palestine in 1928 (14), first on sweet lime rootstocks grafted to sweet orange, and later on almost every citrus variety when grafted on sweet lime (7, 10, 13, 16). In some species, namely Nacottee tangelo and various mandarin trees, a similar disorder has also appeared in the scion. On these latter varieties, the disease is accompanied by gum impregnation occurring in the bark; this was regarded by us to be a result of varietal response of mandarins and mandarin hybrids to xyloporosis and therefore considered as being affected by xyloporosis (7, 9). Recently the incidence of this type of gummy xyloporosis has greatly increased. Many mandarin varieties, for example, Clementine, Dancy, Youssouf Effendi, of various ages, were found affected.

In 1950 Childs (1) reported the cachexia disease of Orlando tangelo trees in Florida. He considered this gum-impregnate pit-peg disorder to be identical with xyloporosis in Israel.

Childs' (2) transmission experiments confirmed the opinion that the two disorders were identical and priority was given to the name xyloporosis. In his experiments, Orlando tangelo seedlings, grafted with budwood taken from xyloporosis-affected sweet orange tree on sweet lime rootstock, produced pitting of the cachexia type. On the other hand, sweet lime seedlings, grafted with budwood from cachexia-affected Orlando tangelo, showed characteristic xyloporotic pitting. Olson and Shull (6) and Olson (5) obtained similar results in Texas.

In our small-scale preliminary experiments conducted under insect-proof conditions (12), sweet lime seedlings graft-inoculated with budwood from cachexia-infected Nacottee tangelo produced typical xyloporotic pitting, confirming Childs' and Olson's results.

Recently Grant, et al. (3) challenged the opinion that cachexia and xyloporosis are identical, and presented evidence that the two are distinct disorders. They graft-inoculated 26 Orlando tangelo and 25 sweet lime seedlings with budwood from cachexia-infected trees. Although the Orlando tangelo seedlings produced typical cachexia symptoms, the sweet lime seedlings failed to produce any symptoms.

The results of systematic observations on Clementine trees in Israel are presented here; they may help to clarify this problem.

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[1] Publication of the National and University Institute of Agriculture, Rehovot. 1961 series, No. 374-E.

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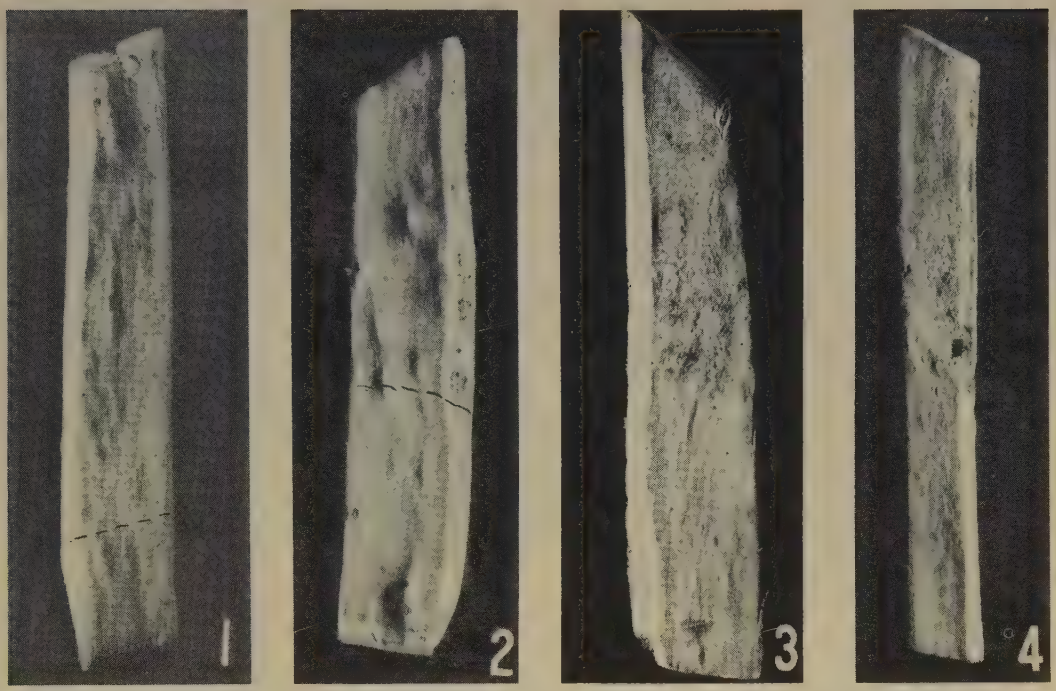


FIGURE 1. Bark sample of Clementine on sweet lime affected by xyloporosis in stock and scion. The dotted line indicates the bud union.

FIGURE 2. Bark sample of Clementine on sweet lime affected by xyloporosis in the stock and by cachexia in the scion. Gum impregnation can be seen in the scion. The dotted line indicates the bud union.

FIGURE 3. Bark sample of Clementine on sweet lime affected by xyloporosis in the stock and by inverse pitting in the scion.

FIGURE 4. Bark sample of Clementine on sweet lime affected by xyloporosis in the stock and by cachexia + inverse pitting in the scion. Gum impregnation without the usual pegs can be seen in the scion.

## METHODS

Observations were made in the summer of 1960 on 15-year-old Clementine trees in a single orchard at Kefar Yona in the Coastal Plain. One plot consisted of 200 trees grafted on sweet lime, and the second plot of 22 trees grafted on sour orange rootstocks.

The trees of both plots grew in a loamy soil and had overhead irrigation. The source of budwood could not be traced.

Each individual tree was examined for its general state of health. In addition, a piece of bark was stripped off across the bud union to detect any possible pathological symptoms in the cambium region of the stock and scion.

## RESULTS

A. Grafted on Sweet Lime: The results presented in Table 1 show that three types of pitting were observed on Clementine trees when grafted on sweet lime rootstock: 1) xyloporotic type (Fig. 1), 2) cachexia type (Fig. 2), and 3) inverse pitting (Fig. 3). The cachexia type was characterized by the presence of gum pockets in addition to the xyloporotic type pegs in the bark. The inverse pitting was characterized by tiny pits on the inner surface of the bark and corresponding pin-point-like outgrowths in the wood. But, bark specimens from some of the trees affected by inverse pitting in the scion exhibited also gum pockets (Fig. 4) and were therefore classified as cachexia infected (Table 1, Group 4).

The infected trees displayed various foliage conditions -- no decline, moderate decline, and severe decline.

The trees were classified into eight groups according to the presence of various combina-

Table 1. Observations on 200 Clementine trees (Groups 1 - 8) on sweet lime rootstocks.

| Group | Pitting symptoms: stock | Pitting symptoms: scion | Occurrence of disorders: no. of trees (per group) | Occurrence of disorders: % of trees | Appearance of trees (%): normal | Appearance of trees (%): moderately declined | Appearance of trees (%): severely declined |
|---|---|---|---|---|---|---|---|
| 1 | Xyloporosis | Cachexia | 95 | 47.5 | 29 | 47 | 24 |
| 2 | Xyloporosis | Xyloporosis | 40 | 20 | 67.5 | 25 | 7.5 |
| 3 | Xyloporosis | None | 39 | 19.5 | 69 | 24 | 7 |
| 4 | Xyloporosis | Cachexia + Inverse pitting | 15 | 7.5 | -- | 20 | 80 |
| 5 | Xyloporosis | Xyloporosis + Inverse pitting | 3 | 1.5 | -- | 33.3 | 66.6 |
| 6 | Xyloporosis + Inverse pitting | Inverse pitting | 1 | 0.5 | -- | -- | 100 |
| 7 | Inverse pitting | Inverse pitting | 1 | 0.5 | -- | -- | 100 |
| 8 | None | None | 6 | 3 | 100 | -- | -- |

tions of pitting types in the stocks and scions: 1) xyloporosis in the rootstock and cachexia in the scion; 2) xyloporosis in the rootstock and in the scion; 3) xyloporosis in the rootstock and smooth scion; 4) xyloporosis in the rootstock, and cachexia intermixed with inverse pitting in the scion; 5) xyloporosis in the rootstock, and xyloporosis intermixed with inverse pitting in the scion; 6) xyloporosis intermixed with inverse pitting in the rootstock and inverse pitting in the scion; 7) inverse pitting in the rootstock and in the scion; 8) rootstock and scion free of any symptoms.

The results in Table 1 demonstrate that the cachexia type of pitting affecting the scion when grafted on sweet lime had a far greater declining effect on Clementine trees than the xyloporosis type. This can be inferred from two facts: 1) The high percentage of occurrence of cachexia on the scions of Clementine trees -- 47.5%, as compared with 20% of the xyloporosis-affected scions, and 2) the severity of decline caused by cachexia -- 24% of cachexia-affected trees showed severe top decline as compared with only 7.5% of the xyloporosis-affected trees.

The results also show that the xyloporosis type of pitting, when it occurs on the scion of Clementine trees, has no effect, either on the number of affected trees or on their state of decline: 1) the percentage of trees showing xyloporosis on both stock and scion equalled the percentage of trees showing xyloporosis on the stock only, that is, 20% versus 19.5%; 2) of the trees affected by xyloporosis in both components, 7.5% showed severe decline, and of those in which only the rootstock was affected -- 7%. Thus the additional infection of the scion by xyloporosis did not aggravate the state of decline of the trees.

The percentage of trees showing other pathological features (inverse pitting alone or in combination with xyloporosis or cachexia) was lower than the two other types alone. In all cases where inverse pitting occurred, either alone or in association with other types of pitting in the scion, the trees showed stronger top decline from 66.6 to 100% versus 24% and 7.5% for cachexia and xyloporosis, respectively, thus demonstrating the severe declining effect of inverse pitting (Fig. 5).

Only 3% of the trees examined were free from any pitting symptoms. These trees showed no decline and were nicely developed.

B. Grafted on Sour Orange: The results of observations on trees grafted on sour orange rootstocks appear in Table 2. All trees, without exception, displayed the same morbid symptoms: cachexia in the cambium region of the scion. No pitting of any type could be detected on the rootstock (Fig. 6). Of the trees examined, 55% showed moderate top decline (Fig. 7). No case of severe decline was observed. This percentage is lower than in trees grafted on sweet lime -- in which 71% (47 + 24) showed decline. This was brought about by the additional infection of the sweet lime rootstock by xyloporosis.

## DISCUSSION AND CONCLUSIONS

The results of our survey on Clementine trees challenge the opinion that xyloporosis and cachexia are caused by the same virus. In our case, trees of the same variety, age and growing under similar conditions, displayed dif-

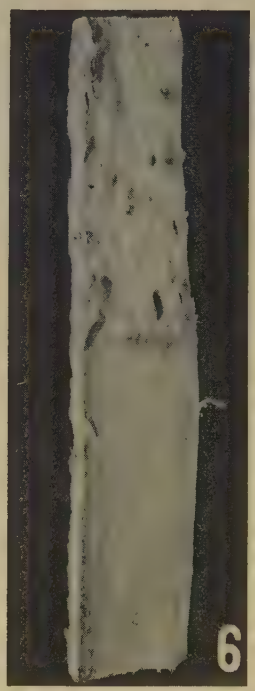

FIGURE 5. Severely declined Clementine on sweet lime affected by xyloporosis in the stock and by cachexia + inverse pitting in the scion.

FIGURE 6. Bark sample of Clementine on sour orange affected by cachexia in the scion. The stock is free of any symptoms.

FIGURE 7. Moderately declined Clementine on sour orange affected by cachexia.

Table 2. Observations on 22 Clementine trees on sour orange rootstocks.

| Pitting symptoms | | Occurrence of disorder | | Appearance of trees (%) | | |
|---|---|---|---|---|---|---|
| stock | scion | no. of trees | % of trees | normal | moderately declined | severely declined |
| None | Cachexia | 22 | 100 | 45 | 55 | -- |

ferent symptoms: one typical for cachexia and the other typical for xyloporosis. This suggests that both xyloporosis and cachexia may be involved in the decline of Clementine trees, each presenting a distinct virus disorder.

The contradictory results obtained by Grant, Childs and others in their transmission experiments can only be explained by the assumption that the budwood source used by Grant car-

ried only the cachexia virus, whereas that used by Childs and others carried both the xyloporosis and cachexia viruses.

The third disorder, inverse pitting, has long been found in Israel on various citrus varieties affected by xyloporosis (8, 10, 15, 16). All such trees showed a more severe decline than those showing only xyloporotic pitting. Inverse pitting on the scion has also been found to increase the decline effect of tristeza-affected trees. The severe decline of tristeza-affected Ellendale mandarin, accompanied by inverse pitting in the scion (11), cannot be ascribed to tristeza alone, as it was grafted on Cleopatra tristeza-tolerant rootstock. It can be explained, however, if the additional disorder, inverse pitting, is taken into consideration.

Our observations on Clementine trees bear out the assumption that inverse pitting might be a separate disorder aggravating the decline of trees affected by other virus diseases. The trees affected by cachexia associated with inverse pitting demonstrated a higher percentage of severely declining trees than those affected by cachexia alone, 80% versus 24%. In trees affected by xyloporosis associated with inverse pitting, the virulence of the inverse pitting was still greater -- 66.6% compared with only 7.5% for trees affected by xyloporosis alone. This assumption is strengthened by the fact that two of the severely declined trees displayed, in their scions, inverse pitting alone.

Among the trees affected by xyloporosis or cachexia, a number of trees retained their normal appearance, whereas no normal tree was found among the 20 trees affected by inverse pitting alone or associated with other disorders.

Similar observations on the parasitic effect of inverse pitting, when associated with cachexia, have been reported recently by Knorr and Price (4). They described a decline of Murcott tangerines in Florida, and called this pathological appearance fovea. These investigators adopted a working hypothesis that the decline of Murcott tangerines is caused by cachexia, and the inverse pitting may be a varietal response to it. Yet they left open the possibility that the decline may be caused by another virus which results in inverse pitting.

Our results seem to prove that inverse pitting may be a separate pathological disorder. If this is so then it is reasonable to assume that the decline of Murcott trees is also caused by two pathogenic factors, cachexia and inverse pitting. Decisive proof of whether inverse pitting represents a separate pathogen can only be obtained by exact transmission experiments.

The conclusions to be inferred from observations on Clementine decline in Israel, and from observations in Florida, are: 1) mandarins may be threatened by three separate diseases attacking the scion: cachexia, xyloporosis and inverse pitting; 2) inverse pitting occurring either alone or associated with cachexia or xyloporosis greatly aggravates the decline of trees; 3) Clementine trees grafted on sweet lime decline more severely than those grafted on sour orange, as the latter is more resistant to xyloporosis than the former.

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# INFLUENCE OF HOST PASSAGE ON VIRULENCE OF PHYTOPHTHORA PARASITICA VAR. NICOTIANAE

P. D. Dukes and J. L. Apple

## Abstract

Two isolates of Phytophthora parasitica var. nicotianae derived from single zoospores were passed serially through eight black shank susceptible (Bottom Special) and black shank resistant (Coker 139) tobacco plants. Each successive series of plants was inoculated with diseased tissue from the preceding series and without re-isolation of the fungus. Reisolates were obtained, however, from the fourth and eighth series plants. Comparative pathogenicity tests of the resulting isolates were made using the same resistant and susceptible tobacco varieties. The virulence level of both isolates was increased significantly by eight serial passages through plants of the susceptible and resistant varieties. Only one isolate was increased in virulence after the fourth passage through the susceptible variety and neither isolate showed a significant increase in virulence after the fourth passage through the resistant variety. This study demonstrates the "plasticity" of this organism with respect to level of virulence and suggests that, under field conditions, more highly virulent biotypes may arise as a result of continuous tobacco culture in infested soil with either susceptible or resistant varieties.

## INTRODUCTION

The long-range success or stability of a plant disease control program based on host resistance or immunity is, to a large degree, dependent upon the inherent and potential pathogenic variability of the causal organism involved. The black shank disease of tobacco is controlled chiefly by the use of resistant varieties in North Carolina and in many other States. The natural population of the causal organism, Phytophthora parasitica (Dast.) var. nicotianae (Breda de Haan) Tucker, is comprised of many strains that vary in virulence from low to very high even on moderately resistant varieties (1). Varieties that were once considered moderately resistant are now classed as having low resistance. There is little reason to question the genetic stability of the varieties, consequently, a change in the virulence of the pathogen may be involved.

Changes in the pathogenicity and/or virulence of fungi resulting from serial host passage have been reported by numerous investigators. Within the genus Phytophthora, Reddick and Mills (8) reported increased virulence of P. infestans resulting from serial passage through plants of a resistant variety. The isolate used apparently came from a single zoospore; however, this point is not clear from their paper. Mills (7) increased the virulence of single zoospore isolates from potato by seven serial passages through tomato foliage. In an extensive study, de Bruyn (4) confirmed the work of Mills (7). Isolates of the tomato race and potato race 1 derived from single zoospores were changed to potato race 2 by serial passage on the appropriate potato host, and potato races 1 and 2 were changed to the tomato race as a result of passage through tomato. This apparent pathogenic instability and innate capacity of this organism to acquire a new pathogenic and/or virulence potential has been termed "plasticity" by Reddick and Mills (8). Results obtained by Black (3) and others agree with those of Mills and de Bruyn.

These observations have formed the basis for the theory that virulence can be altered in P. infestans by passages through "resistant" plants and that the newly acquired characteristic does not change during further cultivation on the same host variety. Others (9) have proposed, however, that the phenomenon of adaptation in P. infestans is nothing more than natural selection within a population of mixed races.

The objective of the present study was to determine the influence of parallel serial passage through resistant and susceptible host plants on the virulence of P. parasitica var. nicotianae.

## METHODS AND MATERIALS

Two virulent isolates of P. parasitica var. nicotianae, one a flue-cured (1030) and the other a burley strain (1156), were passed serially through plants of the varieties Bottom Special

Table 1. Comparative pathogenicity indices of isolates of P. parasitica var. nicotianae after passage through susceptible and resistant tobacco plants[a].

| Isolate[b] | Pathogenicity index by variety[c] | | |
|---|---|---|---|
| | Bottom Special | Coker 139 | Isolate mean |
| 1030 - 0 | 4.1 | 3.0 | 3.6 |
| -S4 | 5.3 | 4.0 | 4.6 |
| -S8 | 6.3 | 4.3 | 5.3 |
| -R4 | 4.5 | 2.9 | 3.7 |
| -R8 | 6.3 | 5.6 | 5.9 |
| 1156 - 0 | 6.1 | 4.4 | 5.2 |
| -S4 | 6.1 | 4.1 | 5.1 |
| -S8 | 6.7 | 5.9 | 6.3 |
| -R4 | 5.4 | 4.2 | 4.8 |
| -R8 | 7.3 | 5.4 | 6.4 |

[a]Combined data from two experiments. For method of computing pathogenicity index, see Apple (1).
[b]Isolate designations as follows: 1030-0 = parental isolate from single zoospore; -S4 = reisolate of 1030-0 after fourth passage through susceptible host; -S8 = reisolate of 1030-0 after eighth passage through susceptible host; -R4 = reisolate of 1030-0 after fourth passage through resistant host; -R8 = reisolate of 1030-0 after eighth passage through resistant host. The designation for isolates from 1156 are the same.
[c]LSD between isolate means: 5% = 0.9; 1% = 1.2. LSD between two variety means with same isolate: 5% = 1.7; 1% = 2.2. LSD between two isolates on same variety: 5% = 1.5; 1% = 2.0.

(susceptible) and Coker 139(resistant). Initially cultures of each isolate were obtained from a single zoospore and maintained on oatmeal agar slants at 10° C without being transferred for the duration of the study.

The roots of plants grown in steamed soil in 4-inch pots were wounded and inoculated with a mycelial suspension of the fungus when the plants were 6 inches high. Infected tissue from the first plants in each group that became diseased was used to inoculate the next group of plants by inserting slivers of diseased stem tissue into the root zone. The time required to complete one serial passage varied from 2 to 3 weeks for the susceptible variety and 4 to 8 weeks for the resistant variety.

Isolations of the fungus were made from the fourth and eighth series of plants. Those obtained after the fourth passage were also stored at 10° C in sealed test tubes to minimize the opportunity for genetic change.

Pathogenicity comparisons were made between the original single spore isolates of the two strains and those from the fourth and eighth passage series using the seedling inoculation test as described by Apple (1). With this technique, nine seedlings of either the Coker 139 or Bottom Special variety were transplanted to each 8 x 8 x 2-inch aluminum tray containing soil and inoculated when in the 4- to 5-leaf stage.

A split-plot experimental design was used with isolates as the whole-plot variable and varieties as sub-plots. Thus, each replicate consisted of one tray each of Bottom Special and Coker 139. Five replicates were used in each of two experiments. Disease readings, beginning with the third day after inoculation and ending 2 weeks later, were used to compute a pathogenicity index for each isolate (1).

## RESULTS AND DISCUSSION

The virulence level of isolate 1030 was increased significantly by four and eight serial passages through plants of the susceptible variety Bottom Special and by eight serial passages through plants (Fig. 1A) of the resistant variety, Coker 139 (Isolate means, Table 1). The virulence level of isolate 1156 was increased significantly by eight serial passages through either a susceptible or a resistant host (Fig. 1B). The virulence of 1030 was unchanged after the fourth passage (1030-R4) through the resistant host. Likewise, isolate 1156 was unchanged after the fourth passage through susceptible (1156-S4) or resistant (1156-R4) plants.

The stock cultures used in this study were derived from single zoospores, therefore, it

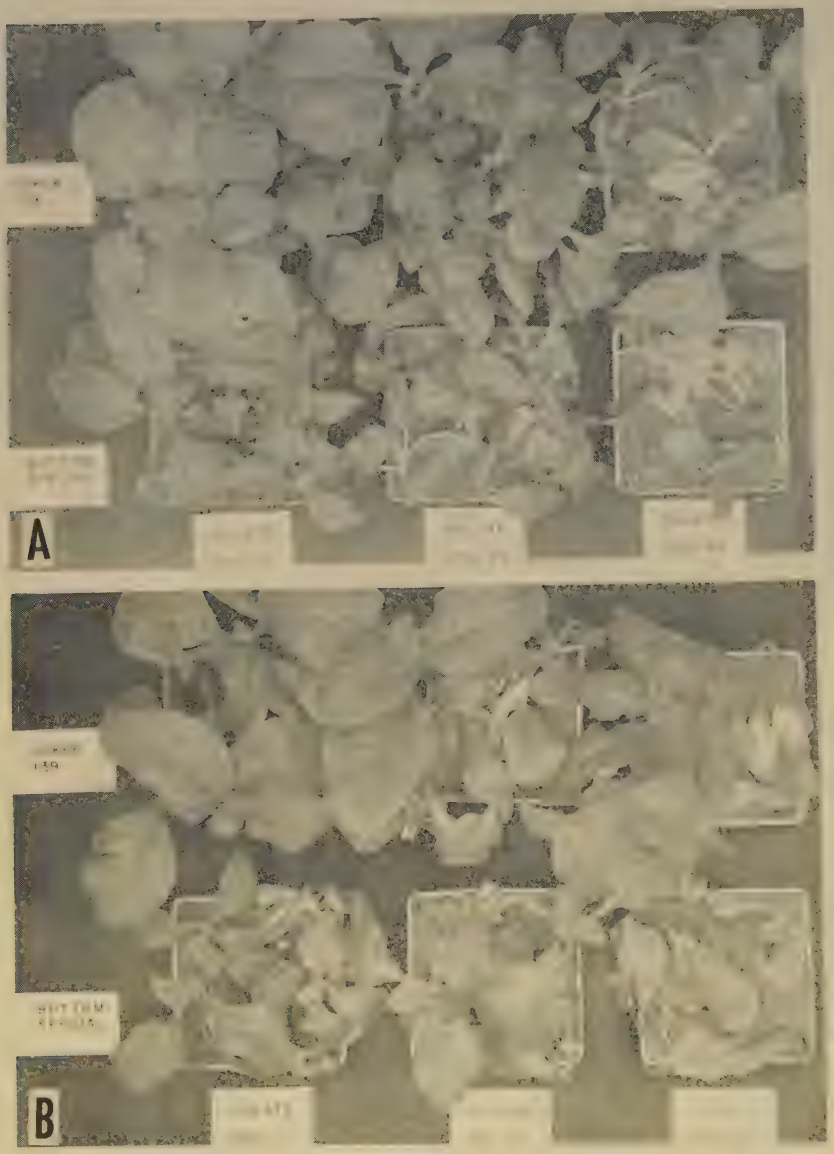


FIGURE 1. Effect of serial host passage on virulence of P. parasitica var. nicotianae as shown on plants of varieties Coker 139 (resistant) and Bottom Special (susceptible). A -- Isolate 1030: 1030-0 = original single zoospore isolate; 1030-R4 = reisolate after fourth passage through Coker 139; 1030-R8 = reisolate after eighth passage through Coker 139. B -- Isolate 1156: 1156-0 = original single zoospore isolate; 1156-R4 = reisolate after fourth passage through Coker 139; 1156-R8 = reisolate after eighth passage through Coker 139.

was assumed that they were derived from a single haploid nucleus (6). Consequently, it is difficult to explain the observed increase in virulence on a genetic basis. More highly virulent mutants could have evolved, in which case they would have been perpetuated by the strong selection pressure imposed; however, this does not appear a highly plausible explanation. This phenomenon could be explained on the basis of adaptive enzyme systems, but no direct evidence is available to support this possibility. It could also be explained on the basis of changes in the zoosporangial production potential or an increase in the motility period of the zoospores which are positively correlated with virulence (5). If it were possible to determine the genetic basis of pathogenicity in the black shank fungus (2), one would be in a better position to explain the observed increase in virulence. Regardless of the mechanism involved, whether it is due to mutation and subsequent selection or adaptation in lieu of genetic change, these studies illustrate the "plasticity" of the black shank fungus with respect to virulence. It is known that the original cultures were not composed of mixed biotypes as has been suggested by Stakman and Harrar (9) as an explanation for similar results obtained with P. infestans. Since Mills (7) and de Bruyn (4) utilized cultures from single zoospores, it is also unlikely that their results reflected selection within a population of mixed races.

One possible limitation in this study was the fact that the stock cultures were maintained for 8 months on artificial media prior to the comparative pathogenicity tests. While in storage,

these isolates could have become less virulent, as has been observed by previous investigators. However, they were stored at 10° C in large test tubes (25 mm diameter) sealed with aluminum foil and were not subcultured during this period. This offered a minimum opportunity for weakly pathogenic mutants to arise since vegetative reproduction was very low.

This study suggests that, under field conditions, more highly virulent biotypes may evolve as a result of continuous tobacco culture in infested soil with either susceptible or resistant varieties. The fact that some resistant varieties are not so effective now in controlling the disease as when they were released lends support to this hypothesis. This could offer also an explanation for the observations of Apple (1) that infested fields planted to a resistant variety for 2 to 3 years yielded a higher percentage of highly virulent biotypes than fields with no resistant variety history. He did not contend that the virulence level was increased by host passage, but that the resistant host merely selected out and perpetuated the highly virulent types that occur in a natural population. Both phenomena may be involved. Therefore, future tobacco varieties may require a higher level of resistance to achieve economic disease control.

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# SYSTEMIC CONTROL OF POWDERY MILDEW OF ROSES (SPHAEROTHECA PANNOSA) WITH THE SEMICARBAZONE DERIVATIVE OF ACTI-DIONE[1]

Brian M. Jones and H. G. Swartwout

The semicarbazone derivative of Acti-dione (cycloheximide)[2] was evaluated for systemic action against powdery mildew of roses. Other workers have shown this derivative to be systemically active against cherry leaf spot (Coccomyces hiemalis) (3, 4, 5) and stem rust of wheat (Puccinia graminis var. tritici) (1, 2, 6).

Soil drenches of the semicarbazone derivative were applied to young, own-root plants of the Snow White variety of roses grown in 6-inch pots. Concentrations used were 2.5, 5, 10, 20, 40, 80 and 100 ppm in aqueous solution. Two hundred milliliters of each concentration were applied to each of three plants. The actual amounts of the derivative applied per pot for the respective concentrations were 0.5, 1, 2, 4, 8, 16 and 20 milligrams. Three days were allowed for up-take of the derivative; the plants were then inoculated with powdery mildew spores.

When disease development was noted on untreated checks, intensity of disease incidence was recorded. For each plant, total lesions, total leaflets of susceptible age and infected leaflets were counted. Approximately 100 leaflets were examined for each treatment. Lesions per leaflet and percentage control for each treatment (total of three plants per treatment) gave the comparative data presented in Table 1. Lesions per leaflet for each treatment were calculated by dividing total lesions by total leaflets examined. Percentage control was calculated by subtracting percentage infection from 100. Percentage infection was calculated by dividing the number of lesions per leaflet for the treatment by the number of lesions per leaflet for the check and multiplying this quotient by 100.

Table 1. Incidence of powdery mildew[a] on Snow White roses treated with soil drenches of the semicarbazone derivative of Acti-dione.

| Concentration (ppm) | Lesions per leaflet[b] | % control[c] |
|---|---|---|
| 0 (check) | 3.60 | 0 |
| 2.5 | 0.58 | 83.89 |
| 5 | 1.33 | 63.06 |
| 10 | 0.04 | 98.89 |
| 20 | 0 | 100 |
| 40 | 0 | 100 |
| 80 | 0 | 100 |
| 100 | 0 | 100 |

[a] Composite of three plants for each treatment.

[b] Total lesions divided by total leaflets examined.

[c] Percentage infection (lesions per leaflet for the treatment divided by lesions per leaflet for the check and this quotient multiplied by 100) subtracted from 100.

As shown in Table 1, partial control was obtained at 2.5, 5 and 10 ppm; and complete control was obtained at 20 ppm and higher concentrations. The actual amount of Acti-dione semicarbazone required for complete control was four milligrams per plant. No phytotoxicity was observed at any of the concentrations used.

Systemic control of rose powdery mildew by soil applications of the semicarbazone derivative of Acti-dione suggests the possibility of systemic control of this disease by other derivatives of Acti-dione.

[1] Contribution from the Missouri Agricultural Experiment Station, Journal Series No. 2281. Approved by Director.

[2] Acti-dione semicarbazone was furnished by the Upjohn Company of Kalamazoo, Michigan.

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# CONTROL OF POWDERY MILDEW OF BLUEBERRY

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## Abstract

High incidence of powdery mildew of blueberry, caused by Microsphaera alni DC., occurred in a number of plantations in the Lake Michigan blueberry-growing area. Lesions formed by the characteristic dendritic spots composed of hyphal strands were counted by two methods: 1) leaves were tagged and several counts were made throughout the season and, 2) leaves were picked at random and the lesions counted in the laboratory.

Standard powdery mildew fungicides were tested to determine their potential for control. Sulfur, Karathane, and Acti-dione, in that order, were found to be the most efficient in control.

## INTRODUCTION

Powdery mildew, caused by Microsphaera alni DC., is one of the most common fungal diseases of the highbush blueberry, Vaccinium corymbosum (2, 4). Few attempts have been made to control the disease in Michigan as the damage produced is not easily observed (1). Further, it has been reported as widespread only after harvest (3), but in Michigan it may be found in early July. In recent years mildew has been found in virtually every blueberry plantation by mid-July. The purpose of this investigation was to determine the potential for control by some of the standard mildew fungicides.

## MATERIALS AND METHODS

The Jersey variety of blueberry was used for the fungicide evaluations as it is the major Michigan variety and is extremely susceptible to powdery mildew.

Preliminary (1958) and final tests (1959-1960) were made in plantations with a history of heavy infection. In 1959 the following protective fungicides were tested: 2-(1-methylheptyl)-4,6-dinitrophenyl crotonate(Karathane), wettable sulfur, basic copper chloride and copper sulfate(C-O-C-S),C-O-C-S plus Kolofog (sulfur) plus hydrated lime, 50 wettable N-trichloromethyl thiophthalimide (Phaltan), 3-(2-(3,5-dimethyl-2-oxocyclohexyl)-2-hydroxyethyl)-glutarimide (Acti-dione), and semicarbazone derivative of cycloheximide (Acti-dione liquid concentrate).

Two spreading agents, Niagara Spray Spread and Plyac were also tested at .05%. Each treatment consisted of four bushes with three replications. Seven weekly applications were made beginning in early June, at petal fall. Applications were made at the rate of 300 gallons /acre with hand sprayers.

In 1960 more extensive tests were made with five materials: wettable sulfur (6 pounds/100 gallons), Karathane (3/4 pound/100 gallons) (to each of these materials Niagara Spray Spread was added to make a .05% mixture), and three proprietary antibiotics, Acti-dione (2 ppm plus 20 ounces of PM spreader corrective), Acti-dione (tablet form at 2 ppm plus 4 ounces of Triton B-1956), and semicarbazone derivative of cycloheximide (5 ppm plus 60 ml of Formulation 12, 486-4, as the sticking agent). These materials were applied with a power sprayer adjusted to a fine spray and at 100 pounds' pressure and 300 gallons/acre.

Tests were also made to determine the effect of three eradicants on the incidence of the disease: sodium 4,6-dinitro-o-cresoxide butyl naphthalene sulfonate and sodium chromate (Elgetol) at 2 quarts/100 gallons, phenyl mercuri triethanol ammonium lactate (mercury) at 1 pint/100 gallons, and an oil treated, finely powdered form of calcium cyanamide (Aero Cyanamid dust) at 200 pounds/acre. Three separate tests of soil surface applications were made; a spring application only, a spring plus fall application, and a spring application in conjunction with sulfur spray applied at the same time as the other protectant sprays.

A test to determine the effect of sulfur dust as an aerially applied protectant was made,

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with a greater area being dusted under these conditions. Random areas, however, were chosen and the same number of plants and replications were counted as in the ground-sprayed plots. These plots were dusted four times with 30% sulfur in conjunction with the regular insecticide dusting program of 4% S-(1,2-bis(ethoxycarbonyl)ethyl) O,O-dimethyl phosphorodithioate (malathion) at the rate of 30 pounds/acre.

Each of the protectant treatments was applied to four replications of 21 plants each. Each replication was made up of three rows of seven plants per row.

The eradicant treatments were applied to four replications of 20 plants each; two rows of 10 plants per row with two replications side by side.

All treatments and replications were randomized except that in the eradicant plots two replications were kept together, thus a large block was used to eliminate the effect of secondary infection from the surrounding area.

Yield trials have been initiated and will be continued for the next several seasons to determine the overall effect of powdery mildew control on yield.

To evaluate the relative potential for control, two methods were incorporated. White tags were numbered 1 to 100 for each replication of each treatment. Twenty tags were placed on each of five of the centrally located bushes in each replication. Thus, the surrounding bushes in that replication served as buffers against secondary infection from the surrounding area. This method seems to simulate more closely an entire sprayed field. Tags were placed on the plants so that each plant was well represented, that is, leaves were tagged at the lower, central and upper portions of each plant. This tag method was used to observe the increase in lesions on each individual leaf.

The second method was simply to pick at random 100 leaves per replication from the same plants as those that were tagged, and count the infection sites per leaf in the laboratory. Three counts were made using the tag method: July 14, August 8, and September 3 to 7. Only one count was made, at the same time as the last tag count.

Disease incidence was determined by counting lesions on the lower leaf surface. Each infection site was identified as a dendritic spot with the characteristic hyphal strands making up the branches. It was difficult to distinguish the number of individual infection sites greater than 10 per leaf, particularly in the later counts. Leaves completely covered with the characteristic hyphal strands were assigned a value of 25; 80% covered, a value of 20 and so on down to 10; at 10 or less each infection site could be distinguished and the counts assigned accordingly.

The high incidence of powdery mildew in the Michigan area on the Jersey variety has been suspected for some time. To authenticate such suspicions, counts were made using the leaf pick method in 18 plantations. The plantations ranged from northern Indiana to Holland, Michigan, and the counts were made between September 7 and 9, 1960.

## RESULTS

Results of the 1958 and 1959 tests indicated, 1) a spreading agent is essential in control; 2) sulfur, Karathane, and Acti-dione were superior to either C-O-C-S, Phaltan or C-O-C-S plus Kolofog and hydrated lime; 3) Niagara Spray Spread was selected as the only spreading agent for the 1960 tests because slightly better control was obtained with it, and it was desirable to reduce the number of variables.

Wettable sulfur and Karathane gave better control than other materials tested in both the 1959 and 1960 tests. Results of the 1960 test are indicated in Table 1.

In 1959 both Karathane and wettable sulfur caused some spray injury, manifested as a darkly colored necrotic area of the affected leaves. However, the damage was not extensive and no damage was apparent in 1960.

The 1960 test showed a significant difference between the treatments at the 1% level. There was no significant difference between the replications at the 1% level, but a slight significance at the 5% level. There was no significant difference between the two methods of counting; therefore the leaf pick method is recommended over the tag method, unless the increase in infection on an individual leaf at different times is a necessary component of the data to be collected.

Infection data derived from leaves picked at random from the 18 plantations in the Lake Michigan blueberry-growing area are shown in Table 2, and the location of the plantations in Figure 1. The average number of infection sites per leaf from these plantations was 9.67, while that of the non-treatment check plots was 12.73 and 11.39 per leaf for the tag method and the leaf pick method, respectively.

Table 1. Number of leaves counted and lesions resulting from fungicide tests for blueberry powdery mildew held in the Grand Junction area.

| Treatment | Replication 1 leaves | 1 lesions | 2 leaves | 2 lesions | 3 leaves | 3 lesions | 4 leaves | 4 lesions | Average no. of: leaves per replication | Average no. of: lesions per leaf |
|---|---|---|---|---|---|---|---|---|---|---|
| Tag Method: | | | | | | | | | | |
| No treatment | 98 | 1152 | 81 | 1083 | 71 | 931 | 92 | 1189 | 82.50 | 12.73 |
| Elgetol | 94 | 536 | 96 | 711 | 98 | 895 | 97 | 943 | 96.25 | 8.01 |
| Cyanamid | 95 | 800 | 95 | 946 | 96 | 1273 | 99 | 1012 | 96.25 | 10.47 |
| Mercury | 96 | 769 | 94 | 786 | 95 | 1031 | 98 | 1182 | 95.75 | 9.83 |
| Elgetol + sulfur | 79 | 43 | 94 | 83 | 87 | 58 | 88 | 71 | 87.00 | .73 |
| Cyanamid + sulfur | 95 | 184 | 93 | 158 | 93 | 158 | 95 | 301 | 94.00 | 2.13 |
| Mercury + sulfur | 88 | 54 | 87 | 61 | 97 | 109 | 98 | 98 | 92.50 | .87 |
| Sulfur | 98 | 54 | 96 | 93 | 77 | 45 | 91 | 118 | 90.50 | .85 |
| Karathane | 97 | 147 | 93 | 198 | 94 | 82 | 90 | 135 | 93.50 | 1.50 |
| Acti-dione tablet | 95 | 324 | 97 | 189 | 89 | 315 | 69 | 173 | 87.50 | 2.86 |
| Acti-dione semi-carb. | 90 | 352 | 89 | 315 | 93 | 470 | 88 | 247 | 90.00 | 3.84 |
| Acti-dione + PM | 94 | 217 | 97 | 125 | 95 | 218 | 80 | 176 | 91.50 | 2.01 |
| Sulfur air dust | 84 | 202 | 96 | 320 | 84 | 375 | 86 | 114 | 87.50 | 2.88 |
| Leaf Pick Method: | | | | | | | | | | |
| No treatment | 100[a] | 1051 | 100[a] | 1215 | 100[a] | 1168 | 100[a] | 1125 | 100.00[a] | 11.39 |
| Elgetol | | 636 | | 942 | | 865 | | 1032 | | 8.68 |
| Cyanamid | | 974 | | 948 | | 1304 | | 1158 | | 10.96 |
| Mercury | | 840 | | 1081 | | 865 | | 1054 | | 9.60 |
| Elgetol + sulfur | | 189 | | 109 | | 105 | | 118 | | 1.30 |
| Cyanamid + sulfur | | 219 | | 159 | | 175 | | 348 | | 2.25 |
| Mercury + sulfur | | 58 | | 98 | | 295 | | 139 | | 1.47 |
| Sulfur | | 149 | | 217 | | 144 | | 124 | | 1.58 |
| Karathane | | 244 | | 375 | | 330 | | 213 | | 2.90 |
| Acti-dione tablet | | 595 | | 343 | | 312 | | 282 | | 3.83 |
| Acti-dione semicarb. | | 471 | | 407 | | 619 | | 517 | | 5.03 |
| Acti-dione + PM | | 369 | | 204 | | 407 | | 266 | | 3.11 |
| Sulfur air dust | | 372 | | 275 | | 495 | | 287 | | 3.57 |

[a]100 leaves counted in all cases.

Table 2. Average number of lesions occurring on 100 leaves picked at random from Jersey blueberries grown in 18 different plantations in the Lake Michigan blueberry-growing area.

| North | Central | South |
|---|---|---|
| 16.05 | 17.23 | 8.17 |
| 8.03 | 9.97 | 10.72 |
| 9.61 | 4.11 | 11.10 |
| 15.99 | 5.26 | 9.11 |
| | 16.77 | 6.92 |
| | 12.69 | 2.44[a] |
| | 1.93[a] | |
| | 8.10 | |

[a]Referred to as plantation number 11 and 18 respectively.


FIGURE 1. Location of the 18 plantations where leaves were picked at random to determine incidence of powdery mildew in the Lake Michigan blueberry-growing area.

The eradicant plots showed a limited amount of control; however when combined with sulfur, control was enhanced. This is most likely due to the sulfur since it alone gave good control.

## DISCUSSION

These tests were initiated as a first step in determining the possible overall effect of powdery mildew on subsequent blueberry yields.

Sulfur and Karathane, using a suitable spreader sticker, and Acti-dione plus PM are effective in control. But, aerially applied sulfur dust may well be the most practical means of application, since only four applications were made by air while five wettable sulfur applications were made with ground equipment. Thus, a fifth application of sulfur by air probably would result in even better control.

Apparently secondary infection increases the incidence of the disease at a rapid rate after early August. Application of fungicide at this time undoubtedly would reduce the secondary infection. It is important to apply one application early after petal fall to reduce primary infection and to make further applications throughout June, July and August to reduce secondary infection.

Although there is no significant difference between the two, the tag method is important for observing the increase in infection on an individual leaf. Tags are placed on leaves at a time when primary infection is probably at its highest rate, while leaves counted by the leaf pick method are often younger leaves and are those which have been infected by secondary inoculum.

High incidence of disease in the Jersey variety is indicated in Table 2, although two of the eighteen plantations observed showed a low incidence of the disease. This may have been due to unique conditions in those particular plantations. Plantation No. 11 was planted alternately with one Jersey row and three Rubel rows. Field observations indicate that the Rubel variety

has a much lower incidence of the disease than does Jersey in Michigan. Bergman (1) and Demaree and Wilcox (2) list them both as intermediately susceptible in the east. Under some conditions Rubel was found to be badly infected (1), however this does not appear to be the case in Michigan. Thus, the disease may not build up as rapidly when such varieties are in alternate rows. Further work along these lines is necessary. Plantation No. 18 is rather young and is not yet producing fruit, so the disease may not have had sufficient time to build up. Further conjecture might indicate that a limited number of fungicide applications would hold the disease in check in such a young field.

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# THE RELATION OF PIGMENTATION AND FREE AMINO ACID CONTENT WITH RESISTANCE TO COLLETOTRICHUM LAGENARIUM IN WATERMELONS[1]

Lee A. Hadwiger and Charles V. Hall[2]

## Summary

The rind color pattern on mature fruits of Garrison watermelon exhibited a distinct influence on the virulence of Colletotrichum lagenarium (Pass.) Ell. & Halst. under field conditions at Manhattan, Kansas. Lesions resulting from infection were concentrated primarily on the dark green stripes. The upper cell layers, including the pigmented tissue of the dark green stripes, contained much higher quantities of citrulline and glutamine and only slightly higher quantities of alanine and aspartic acid than the corresponding tissues of the light green stripes. Rind tissue below the dark green layers also contained significantly more citrulline and glutamine than similar tissue below the light green stripes.

## INTRODUCTION

In pursuing the possibility that resistance of cucurbits to Colletotrichum lagenarium (Pass.) Ell. & Halst. is primarily chemical in nature (2, 3) the mature tissues of watermelon rinds were analyzed to determine the free amino acid content. The variety Garrison was utilized advantageously in that a contrasting degree of susceptibility was exhibited (Fig. 1) on the light and dark green stripes of mature fruits when exposed to natural infection of C. lagenarium at Manhattan, Kansas.

## METHODS AND MATERIALS

Free amino acid determinations were made on extracts from separate tissue samples of the watermelon rind. Upper layer extracts were made by removing 10-gram samples of fresh tissue from the epidermis and all pigmented cell layers from areas of each stripe. Lower layer extracts consisted of 10-gram samples from 1/2 inch of the rind tissue immediately below the pigmented layers. The fresh tissue was immediately placed in 95 ml of 80% ethyl alcohol

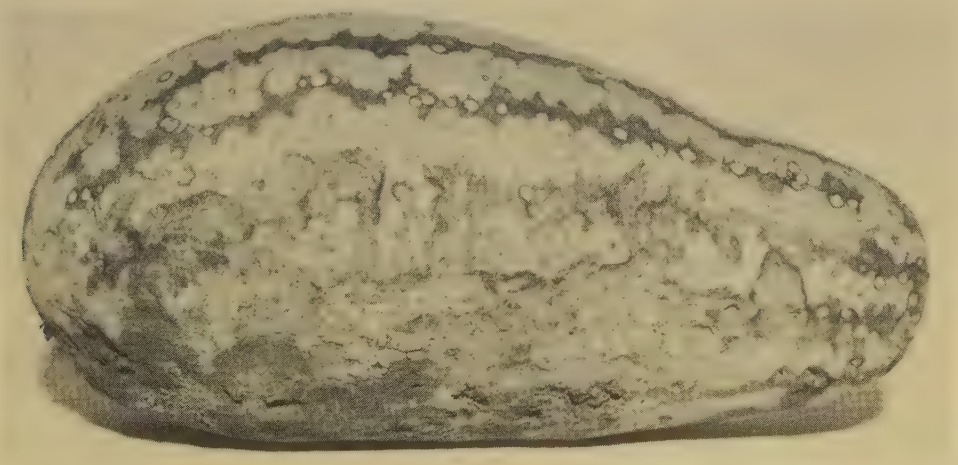

FIGURE 1. Mature fruit of Garrison watermelon showing a differential growth response by C. lagenarium on light and dark green stripes.

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and blended for 3 minutes in a high speed blender. The alcohol extract was filtered and then centrifuged at 30,000 RPMs for 10 minutes. A 60-cc aliquot then was concentrated to a volume of 20 cc by evaporation. A 500-lambda sample was used for spotting on Whatman No. 3 MM 18 1/2 x 22-inch filter paper. Two-way chromatographic separations were made using butanol: water:acetic acid (2.5:2.5:0.6) and water saturated phenol as solvents. A 0.2% solution of ninhydrin in acetone was used for the color indication of amino acid spots. Quantities of amino acids were estimated on the basis of the relative size and density of spots.

## RESULTS

The following amino acids were detected in the watermelon rind tissues: Alanine, arginine, aspartic acid, cysteine, alpha amino butyric acid, citrulline, glutamic acid, glutamine, glycine, histidine, hydroxyproline, isoleucine, leucine, lysine, methionine, proline, serine, threonine, and valine. The upper cell layers of the dark green stripes contained significantly higher quantities of citrulline and glutamine; also slightly more alanine and aspartic acid than the corresponding tissues from the light green stripes. Tissues from layers beneath the dark green stripes contained significantly higher quantities of aspartic acid, glutamic acid, serine, and threonine and a lower content of histidine than was present in the lower cell layers of the light green stripes.

## DISCUSSION

The predominance of lesions resulting from infection by C. lagenarium on or near the dark green stripes of the mature fruits would suggest 1) a physical limitation of fungal penetration on light green stripes, 2) increased synthesis of chemical components within the dark green stripes which are more favorable for fungal infection, or 3) the presence of a chemical inhibitory component in the light green stripes.

The possibility of a morphological barrier was not substantiated by Busch and Walker (1) in view of histological studies of leaf tissue infection. The presence of C. lagenarium resistance in mature resistant watermelons is usually characterized by miniature lesions which penetrate below the epidermal layer but are unable to progress farther. This would tend to eliminate the possibility of the epidermis being a physical barrier.

The higher concentration of a few free amino acids in susceptible rind tissues may indicate a more favorable medium for the infecting organism, especially the higher concentration of citrulline and/or glutamine. However, the differential infection conceivably could be related to: vitamin or nucleic acid content, the presence of an inhibitor, growth regulating substances, or antibiotics, the effects of which must be substantiated or eliminated. All possibilities are presently under investigation.

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# POINSETTIA SCAB - A NEW REPORT FOR PUERTO RICO

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On September 21, 1960, while surveying for plant diseases around the Rio Piedras Experiment Station, the writer noticed a heavy infection of poinsettia scab (Sphaceloma poinsettiae Jenkins & Ruehle) attacking a poinsettia bush (Euphorbia pulcherrima). This is the first report of poinsettia scab in Puerto Rico. Since that date numerous specimens of this disease from scattered areas of Puerto Rico have been brought to the attention of Dr. F. L. Wellman, Head of the Department of Plant Pathology and Botany at the Rio Piedras Experiment Station. Judged by the new reports from widely scattered areas in Puerto Rico and by the extent of injury shown by infected plants, poinsettia scab has probably been on the island for some time.

Poinsettia scab was first discovered (3) in a nursery at Honolulu, Hawaii in November 1939. Its preliminary identification as a Sphaceloma was verified (2, 3) by Anna E. Jenkins of the United States Department of Agriculture in 1941. It was found next near Goulds, Florida (5) in 1940 by Dr. George D. Ruehle of the Sub-Tropical Experiment Station at Homestead, Florida. Since these first reports and verifications, the disease has been reported from Jamaica (1) in 1954 and from Brazil (4) in 1955.

Poinsettia scab attacks both the stems and the leaves of poinsettia, resulting in a dieback of the young stems and distortion and wrinkling of the leaves (Fig. 1). The leaves usually drop prematurely, especially if the petioles are infected. Numerous conspicuous raised lesions or cankers are present on the diseased stems. They are circular to elongate and range from 1 mm to 1 cm or more in length. The cankers sometimes completely encircle the stem, causing the loss of foliage above the girdled areas, and die back from the tip. Lesions on the leaves are smaller than those on the stem and are confined chiefly to the petioles, midribs, and veins.

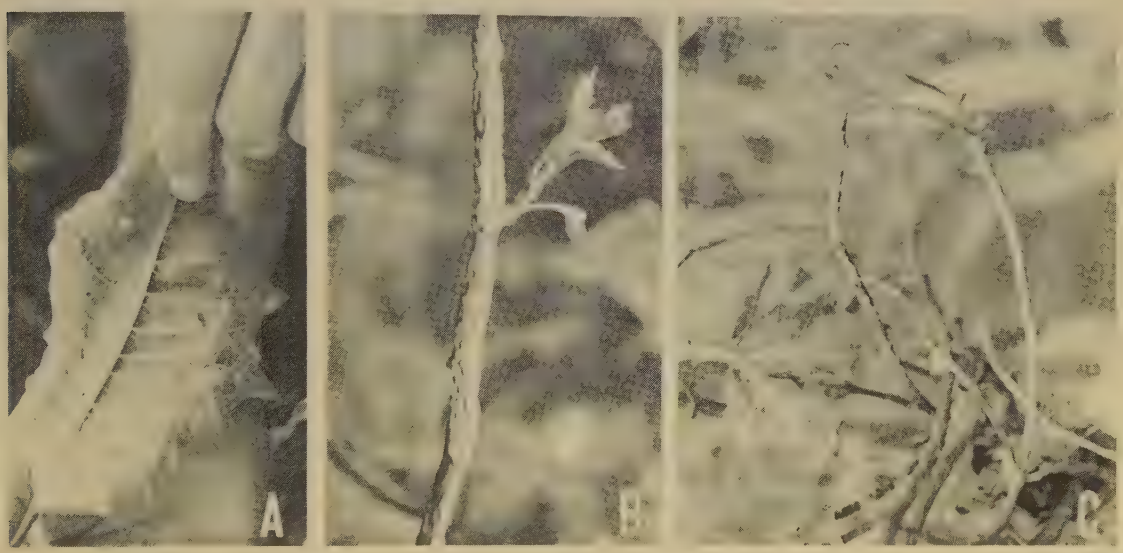


FIGURE 1. Poinsettia scab infection on: A -- leaf; B -- stem; C -- twigs.

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# COMPARATIVE EFFECTS OF SOIL FUNGICIDE TREATMENTS ON SOIL ROT AND DAMPING-OFF OF CUCUMBER[1]

John Paul Jones[2]

## Abstract

Various fungicides and fungicide combinations were applied as pre-seeding and lay-by drenches on cucumbers to evaluate their effect in reducing the incidence of soil rot and damping-off. Consistent decreases of soil rot incidence were obtained with Hercules 3944 or combinations of PCNB with Dexon, captan, and Acti-dione (S). Several of the fungicide treatments effectively controlled Pythium damping-off. Rhizoctonia damping-off occurred only in one test and was controlled by PCNB-containing combinations or Hercules 3944.

## INTRODUCTION

Soil rot is the most destructive cucumber disease in Florida and has essentially eliminated all commercial culture on second-year land. Hence, trials were initiated in 1958 primarily to evaluate the effect of various soil fungicides on the incidence of soil rot and secondarily to evaluate the effects of these fungicides on damping-off incidence.

## MATERIALS AND METHODS

Over a 2-year period (fall 1958 - fall 1960) seven soil fungicide trials were conducted using Ashley cucumbers in randomized block designs with four or five replications. All fungicides were applied as soil drenches to the bed 1 week prior to seeding and at lay-by. The

Table 1. Materials evaluated and their active ingredients.

| Material | Composition |
|---|---|
| Acti-dione (S) | 1.6% emulsifiable concentrate (EC). 2-(3,5-dimethyl-2-oxocyclohexyl)-2-hydroxyethyl) glutarimide semicarbazone. |
| B-1843 | 50% wettable powder (WP). trans-1,2,-bis (n-propyl-sulfonyl) ethylene. |
| BFG-Cu | $CuSO_4$ (5%) + formaldehyde (25%). |
| Captan 50W | 50% WP. captan. N-trichloromethylmercapto-4-cyclohexene-1,2-dicarboximide. |
| CM-19 | 17% phenylphenols and related aryl phenols, 2% octyl and related alkyphenols. |
| CP 15986 | 50% W/v EC. Sodium methoxyethyldithiocarbamate. |
| CP 30249 | 50% W/v EC. 2-chloro-3-(tolysulfonyl)-propionitrile. |
| D-113 | 50% WP. 1,2, dichloro-1-(methylsulfonyl) ethylene. |
| Dexon | 70% WP. p-dimethylaminobenzenediazo sodium sulfonate. |
| Dexon-PCNB | WP. 35% Dexon-35% pentachloronitrobenzene. |
| GC 2466 | 50% WP. bis-(3,4-dichloro-2(5)-furanonyl) ether. |
| Hercules 3944 | 50% WP. 5-chloro-4-phenyl-1, 2-dithiol-3-one. |
| K6 | 8.0% solution of 2,2'-methylenebis (3,4,6-trichlorophenol) in an organic solvent. |
| Mylone | 85% WP. 3,5-dimethyltetrahydro-1,3,5, 2H-thiadiazine-2-thione. |
| Nabac 25 | 25% WP. 2,2'-methylenebis (3,4,6-trichlorophenol). |
| Norwich U587 | Organic sulphur compound. |
| Omadine manganese | 50% WP. 2-pyridinethione 1-oxide manganese salt. |
| Omadine zinc | 50% WP. 2-pyridinethione 1-oxide zinc salt. |
| Ortho Phaltan | 50% WP. N-trichloromethylthiophthalimide. |
| Panogen soil drench | 3.5% EC., methylmercury hydroxide. |
| Phygon XL | 50% WP. dichlone (2,3-dichloro-1,4-naphthoquinone). |
| PCNB | 75% WP. pentachloronitrobenzene. |
| Thylate | 55% WP. thiram (tetramethylthiuram disulfide). |

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Table 2. Ashley cucumber fruit yields, final stands, and percentages of soil rotted fruit as affected by various chemical soil drenches applied to the bed 1 week prior to seeding and at lay-by.

| Treatment | Pounds/acre | % final stand[a] | No. marketable fruit | % soil rot |
|---|---|---|---|---|
| | | Trial 1 (fall 1958) | | |
| Captan | 40 | 70.1 | 297 | 30.0 |
| PCNB | 30 | 35.3 | 202 | 16.0 |
| PCNB | 45 | 30.0 | 168 | 7.6 |
| PCNB + Acti-dione (S) | 30 + 58 ppm | 23.8 | 204 | 9.8 |
| Captan + Acti-dione (S) | 40 + 50 ppm | 88.4 | 310 | 43.2 |
| Acti-dione (S) | 58 ppm | 70.0 | 275 | 23.3 |
| Nabac 25 | 2 | -- | 171 | 21.6 |
| PCNB + captan | 30 + 40 | 55.6 | 232 | 14.2 |
| Check | | 61.2 | 320 | 26.2 |
| LSD .05 | | 10.81 | -- | 5.58 |
| | | Trial 2 (spring 1959) | | |
| PCNB | 75 | 46.5 | 170 | 37.6 |
| Acti-dione (S) | 100 ppm | 68.1 | 409 | 46.2 |
| Captan | 50 | 80.0 | 399 | 33.6 |
| Captan + Acti-dione (S) | 50 + 100 ppm | 83.6 | 420 | 41.2 |
| Captan + PCNB | 50 + 75 | 71.3 | 285 | 30.2 |
| Acti-dione (S) + PCNB | 100 ppm + 75 | 57.6 | 251 | 25.9 |
| BFG-Cu | 4900 ppm | 69.5 | 357 | 44.3 |
| Nabac 25 | 6 | 80.1 | 355 | 43.1 |
| CM-19 | 50000 ppm | 76.0 | 296 | 49.7 |
| D-113 | 50 | 76.5 | 298 | 33.9 |
| Dexon | 50 | 71.3 | 345 | 45.2 |
| Phaltan | 50 | 53.5 | 349 | 29.2 |
| Check | | 54.6 | 322 | 48.8 |
| LSD .05 | | 5.55 | 59.7 | 10.2 |
| | | Trial 3 (fall 1959) | | |
| Omadine manganese | 40 | 80.4 | plants killed by frost | |
| Mylone | 200 | 54.0 | | |
| Bayer 1843 | 50 | 62.8 | | |
| Phygon | 100 | 61.9 | | |
| CP 15986 | 50 | 59.8 | | |
| CP 30249 | 20 | 72.2 | | |
| Panogen soil drench | 100 ppm | 57.3 | | |
| Phaltan | 50 | 58.9 | | |
| Thylate | 50 | 48.2 | | |
| Thylate + captan | 50 + 50 | 62.6 | | |
| Check | | 52.8 | | |
| LSD .05 | | 13.22 | | |
| | | Trial 4 (spring 1960) | | |
| Omadine zinc | 100 | 65.6 | 850 | 13.8 |
| Panogen soil drench | 2200 ppm | 73.0 | -- | -- |
| K6 + zinc sulfate | 3 qt. + 0.5 | 84.3 | 878 | 10.5 |
| Norwich U587 | 40 | 76.8 | 651 | 10.8 |
| CP 30249 | 40 | 91.1 | 485 | 5.8 |
| Dexon-PCNB | 75 | 73.7 | 812 | 4.4 |
| Nabac 25 + captan | 5 + 25 | 75.5 | 938 | 8.4 |
| Captan | 25 | 80.1 | 849 | 6.6 |
| Check | | 76.4 | 772 | 20.6 |
| LSD .05 | | 9.88 | 66.1 | 3.45 |

Table 2. (Continued)

| Treatment | Pounds/acre | % final stand[a] | No. marketable fruit | % soil rot |
|---|---|---|---|---|
| | Trial 5 (spring 1960) | | | |
| Omadine zinc | 50 | 73.0 | 698 | 9.9 |
| CP 30249 | 20 | 80.4 | 518[b] | 4.5 |
| Panogen soil drench | 1100 ppm | 79.4 | -- | -- |
| Nabac 25 + captan | 1 + 5 | 79.3 | 627 | 8.9 |
| GC 2466 | 100 | 58.0 | 681 | 9.8 |
| Norwich U587 | 200 | 79.2 | -- | -- |
| Hercules 3944 | 75 | 85.3 | 620 | 5.3 |
| Nabac 25 | 1 | 76.8 | 662 | 10.9 |
| Check | | 76.5 | 682 | 16.3 |
| LSD .05 | | 8.4 | NSD | 4.87 |
| | Trial 6 (fall 1960) | | | |
| Hercules 3944 | 5 | 47.6 | 336 | 14.7 |
| Hercules 3944 | 15 | 55.8 | 404 | 9.4 |
| Hercules 3944 | 25 | 55.2 | 353 | 8.1 |
| Omadine manganese | 50 | 51.3 | 425 | 13.6 |
| Omadine manganese | 25 | 55.1 | 418 | 17.2 |
| Dexon-PCNB | 75 | 73.6 | 299 | 8.0 |
| Dexon-PCNB | 25 | 55.0 | 380 | 15.0 |
| Plastic | | 47.8 | 326 | 13.8 |
| CP 30249 | 20 | -- | 361 | 15.8 |
| Dexon-PCNB | 50 | 69.4 | 418 | 9.3 |
| Hercules 3944[c] | 2 | 62.7 | 414 | 16.9 |
| CP 30249 + captan + PCNB | 10 + 50 + 50 | 73.1 | 400 | 7.2 |
| Hercules 3944 | 50 | 76.9 | 418 | 6.8 |
| Check | | 55.3 | 406 | 17.6 |
| LSD .05 | | 10.7 | NSD | 5.1 |
| | Trial 7 (fall 1960) | | | |
| Plastic | | | 586 | 10.8 |
| Hercules 3944 | 10 | | 602 | 9.2 |
| Hercules 3944 | 15 | | 586 | 10.3 |
| Hercules 3944 | 20 | | 577 | 8.0 |
| Dexon-PCNB | 75 | | 475 | 6.7 |
| Dexon-PCNB | 50 | | 602 | 8.5 |
| Captan + PCNB | 80 + 80 | | 551 | 4.2 |
| Hercules 3944 | 5 | | 605 | 11.3 |
| Omadine manganese | 40 | | 624 | 7.0 |
| Omadine manganese | 25 | | 628 | 9.4 |
| Check | -- | | 597 | 10.4 |
| LSD .05 | | | NSD | NSD |

[a]Damping-off in trials 1, 2, 3, 4, and 5 caused by Pythium sp. and in trial 6 by Rhizoctonia solani.

[b]Although an analysis of variance did not indicate differences among treatments, a comparison of fruit yields from CP 30249 treated plants versus all other yields demonstrated that yields from CP 30249 treated plants were lower than all other yields taken collectively.

[c]In-furrow dust application of Hercules 3944 followed by a CP 30249 lay-by drench.

fungicides used and their rates of application in pounds per acre or parts per million of formulated material are given in Table 1. After final stand counts were taken all plots either were thinned or, in a few instances, partially reseeded in order to obtain uniform stands. Consequently yield differences were due primarily to the lay-by treatments, not to the differences in stands resulting from the pre-seeding drenches.

## RESULTS

Damping-off: Damping-off in trials 1-5 was caused by Pythium sp. and was controlled by several fungicides, the better of which were captan, Acti-dione (S), CM-19, D-113, Omadine manganese, CP 30249, K6 + zinc sulfate, and Hercules 3944 (Table 2). PCNB (Terraclor) and GC 2466 tended to increase the incidence of Pythium damping-off, and in trials 1 and 2 final stands from PCNB + captan and PCNB + Acti-dione (S) plots were less than those from the captan and Acti-dione (S) plots.

Damping-off in trial 6 was caused by Rhizoctonia solani and was effectively controlled by Hercules 3944 at the 50 pounds/acre rate, captan + PCNB, and Dexon-PCNB at the 50 and 75 pounds/acre rates.

Soil Rot: In general, individual fungicides gave no control, inconsistent control, or poor control of soil rot. For example, captan (40 pounds/acre) in trial 1 gave no control, in trial 2 (50 pounds) no to poor control, and in trial 4 (25 pounds) good control. CP 30249 at 40- and 20-pound rates gave good results in trials 4 and 5 but in trial 6 at 20- and 10-pound rates gave no control. PCNB (45 pounds) gave good control in trial 1 but essentially no control at 75 pounds in trial 2.

Hercules 3944, Acti-dione (S) + PCNB, captan + PCNB, and Dexon-PCNB at proper rates gave consistent decreases of soil rotted fruit.

Phytotoxicity: Only two of the trial fungicides, CM-19 and D-113, were phytotoxic when applied as pre-seeding drenches. Seedlings from the CM-19 and D-113 treated plots were chlorotic and slightly stunted. However, after approximately 1 month phytotoxicity symptoms were no longer evident. Lay-by applications with CM-19 and D-113 were not phytotoxic and yields were not decreased. PCNB, CP 30249, Norwich U587, Panogen soil drench, and Dexon-PCNB at high rates were the only materials to decrease yields (Table 2).

## DISCUSSION

Primarily two factors have delayed the development of practical and consistent chemical control measures of cucumber soil rot in Florida: 1) Fungicides cannot be supplied until time of vining (lay-by) and consequently must be non-phytotoxic to cucumber foliage, a restriction which eliminates many fungicides. The extreme cucumber market price fluctuations indirectly dictate that production expenditures be kept minimal. Hence, fungicides cannot be broadcast but must be applied to the bed. Since only the bed is treated no fungicides can be applied, without risk of recontamination, until lay-by, when all cultivation and soil movement from the untreated middles have ceased. 2) Both Rhizoctonia solani Kuehn and Pythium aphanidermatum (Edson) Fitz. must be controlled. Sowell (2) reported that soil rot on the west coast of Florida was caused by R. solani; whereas Walter (3) also from the west coast reported Pythium sp. as the causal agent. Epps (1) stated that cucumber "fruit-rot" in South Carolina was incited by P. aphanidermatum. The author has frequently isolated R. solani and occasionally P. aphanidermatum from diseased fruit, but inoculations of healthy fruit with both fungi separately have resulted in rots unlike typical soil rot. The results of the fungicide trials strongly suggest that soil rot in Florida is caused by both Pythium and Rhizoctonia. The results which indicate such are: 1) captan in two trials did not control soil rot but did in a third trial, 2) PCNB controlled soil rot in one trial but not in another, 3) Hercules 3944, a fungicide which gave damping-off control against both Pythium and Rhizoctonia, gave consistent control of soil rot, and 4) combinations of PCNB, a fungicide known to control Rhizoctonia, with Pythium-controlling fungicides (captan, Acti-dione (S), and Dexon) consistently decreased soil rot. In essence, fungicides that controlled only one of the pathogens, Pythium and Rhizoctonia, did not consistently decrease soil rot; whereas Hercules 3944 or those fungicide combinations which controlled both fungi consistently decreased soil rot incidence.

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# PREPLANT ONLY VERSUS A SECOND TREATMENT ONE YEAR LATER IN THE CONTROL OF ROOT KNOT ON THE PERENNIAL CARYOPTERIS

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## Summary

Preplant treatments with a number of the most effective nematocides now available failed to give effective control of root knot on Caryopteris harvested at the end of the second growing season. One exception involved the use of DBCP, which in that instance was formulated as Nemagon granules (17.3% active), applied at 300 pounds per acre. When DBCP, as Fumazone, was applied as a side-dressing to Caryopteris in the nursery row in June of the second year very good control of root knot was obtained in all four of the plots that received Nemagon at various rates of use as preplant treatments. In a fifth plot treated with Dorlone control was also very good. Wherever the preplant treatment failed to give a fair degree of root-knot control during the first summer, the effectiveness of DBCP as a supplemental treatment in June of the second year was also much less. EN-18133 was comparatively ineffective in the control of root knot when used either as a preplant or a supplemental treatment. Only in the plot where 300 pounds of 17.3% Nemagon granules was followed by Fumazone as a side-dress application in June of the second year were M. hapla larvae less numerous than in the untreated check plot.

It should be noted that DBCP is not effective in the control of root knot in muck soils when applied as a side-dress treatment to rows of vegetable crops.

## INTRODUCTION

Caryopteris incana, a low-growing perennial, is propagated for sale by nurseries in northern Ohio by planting rooted cuttings in July of one year and then digging them in the fall of the second year (about 16 months later). This means they are subject to attack by nematodes during two successive summers. Since Caryopteris is very susceptible to infestation by the root-knot nematode, and many of the fields where it might be grown were known to be heavily infested with Meloidogyne hapla, the effectiveness of various nematocidal formulations as preplant treatments in the control of root knot was tested in a planting made in northern Ohio in the spring of 1959.

## PROCEDURE

Twelve plots, each 30 x 150 feet in size, were treated with the formulations listed in Table 1. All treatments were applied on May 14, 1959 with a tractor-operated rotovator, 60 inches in width, set at a depth of about 8 inches, with soil temperature of 56° F. The soil was a light sandy loam. The residual population of M. hapla was known to be very high following the continuous planting of susceptible host plants in the field during several previous years. The young Caryopteris plants were set in the treated plots during the first week in July, with the rows crossing the treated strips at right angles.

In the fall of 1959, ten plants were selected at random from each of the treated plots and examined for the presence of root knot. At the same time soil samples were taken from the various plots and processed by a modified Baermann funnel technique (1) for the presence of M. hapla larvae. These 1959 data on root-knot infestation and larval populations also are given in Table 1.

In early June of 1960, soil samples were again taken to determine the larval populations at that time. Since they were rather high in some of the plots (table), a portion of the planting was sidedressed with DBCP (Fumazone) and EN-18133 using a single row applicator. The treatments were applied with blades placed 12 inches apart and inserted to a depth of about 10

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Table 1. Comparative control of root knot on Caryopteris with preplant treatments only, and when plants growing in the treated plots were sidedressed 1 year later with two different nematocides. Also the number of M. hapla larvae per pint of soil in fall of first year after treatment, and in June and October of the second year.

| Treatments | Rate per acre | Average root-knot score[a] in plots receiving: Preplant treatments only, October 1959 | Preplant treatments + supplemental side-dressings, June 1960: Fumazone | Preplant treatments + supplemental side-dressings, June 1960: EN-18133 | M. hapla larvae per pint of soil in preplant treatment plots only: Oct. 1959 | June 1960 | Oct. 1960 |
|---|---|---|---|---|---|---|---|
| 1. EDB (731 Em.) | 8 gal. | 0.2 | 1.20 | 3.70 | ---- | 0 | 1060 |
| 2. Nemagon (EC-2) | 6 gal. | 0.2 | 0.13 | 3.20 | ---- | 106 | 2438 |
| 3. D-D (Em.) | 30 gal. | 0.6 | 2.30 | 3.60 | ---- | 800 | 5883 |
| 4. Nemagon (17.3% granules) | 200 lb. | 0.8 | 0.26 | 3.00 | ---- | 160 | 3223 |
| 5. Telone | 30 gal. | 0.6 | 1.90 | 3.50 | 53 | 213 | 3604 |
| 6. EN-18133 | 4 gal. | 1.6 | 3.40 | 3.70 | 586 | 800 | 3127 |
| 7. Nemagon (EC-2) | 8 gal. | 0.2 | 0.07 | 0.30 | ---- | 0 | 2438 |
| 8. None (Check) | ---- | 3.2 | 3.50 | 3.90 | 426 | 1226 | 1908 |
| 9. Dorlone | 24 gal. | 2.0 | 0.30 | 2.20 | ---- | 106 | 3498 |
| 10. Nemagon (17.3% granules) | 300 lb. | 0.0 | 0.00 | 0.30 | 0 | 0 | 106 |
| 11. C-9882 | 5 gal. | 1.0 | 1.00 | 3.80 | ---- | 426 | 1908 |
| 12. D-D | 30 gal. | 2.6 | 1.53 | 4.00 | ---- | 106 | 1855 |

[a]The scale used in scoring the degree of root-knot infestation varied from zero (0) for no root knot to four (4) for the maximum degree present.

EDB = Ethylene dibromide. No. 731 is an emulsifiable formulation, 73% active.

Nemagon EC-2 and Fumazone 70-E = 1,2-dibromo-3-chloropropane. These are emulsifiable formulations at about 50% active.

D-D = 1,3-dichloropropene-1,2-dichloropropane. "Em" is an emulsifiable formulation.

Telone = 1,3-dichloropropene (Technical).

EN-18133 = O, O-diethyl O-2-pyrazinyl phosphorothioate formulated at 4 pounds/gallon.

Dorlone = A mixture of EDB at 18.7% and Telone at 75.2%.

C-9882 = Diphenyl sulfide, 60% emulsifiable concentrate.

inches. Since this method of treatment using these comparatively plant-safe materials had given good control of root knot on Ajuga and Hypericum in the same nursery in 1959 (2), it was used in this experiment in an effort to check the infestation that was beginning to develop. Fumazone 70-E (50% DBCP) was applied at 4, 6, and 8 gallons per acre and EN-18133 (formulated at 4 pounds/gallon) was used at 2 and 3 gallons. On October 18, 1960 the planting was harvested and the plants in the differently treated plots were scored on the basis of the degree of root-knot infestation. Soil samples were also taken from the differently treated plots (preplant treatments) and the larval populations were again determined by the technique mentioned. Nematode populations and degree of root-knot infestation also are given in Table 1.

## RESULTS AND DISCUSSION

Nurserymen searching for a method of controlling nematodes in beds of perennials frequently want to know when the recommended treatment should be applied and how long it will be effective. Definite information is seldom available, but the consensus of opinion is that it will not be effective for more than 2 or 3 years, chiefly because no practical field treatment can ever be thorough enough to eliminate all of the nematodes, and the viable residue may be expected to re-establish an appreciable population within a year or two in the presence of a suitable host.

The data given in the first two columns of Table 1 show that protection against root-knot infestation did not extend even to the second year in these Caryopteris plots. An examination in October 1959 of ten plants from each treatment indicated some control of root knot in every instance when compared with the untreated check, but only Nemagon (treatment 10) at 50 pounds per acre in a granular formulation (500 pounds at 17.3%) gave a zero reading at that time. The degree of infestation varied rather widely with other treatments, ranging from 0.2 (in a scale

graduated from 0 for none to 4 for maximum infestation) for three different treatments, two of which also contained Nemagon, to 2.0 for Dorlone and 2.6 for D-D. Soil samples were taken from only four plots at the time the plant specimens were examined. No root-knot larvae were found where 300 pounds of Nemagon granules were applied (treatment 10) and only a few were present in the Telone plot (treatment 5). Soil treated with EN-18133 (treatment 6) contained an even higher number of root-knot larvae than that of the untreated check plot in October 1959.

Early in June 1960, soil samples were taken from each of the 12 plots in the experiment and their larval populations of M. hapla determined (Table 1, next to last column). Three treatments showed a zero population, although there were obviously some root-knot nematodes present, since none of the three gave zero readings 4 months later. The check plot showed the highest recoverable population at this time, but it was nearly as great in several others.

Previous experience had indicated that root knot could be greatly reduced by treating such perennials as Ajuga and Hypericum with side-dress applications in the nursery row (2). Fumazone (DBCP) and EN-18133 were applied in this manner to several rows of Caryopteris in this experiment on June 17, 1960. Fumazone 70-E was applied at 4, 6, and 8 gallons per acre and EN-18133 at 2 and 3 gallons. The extent to which this second treatment was effective in promoting the control of root knot is indicated in the third and fourth data columns of the table.

The average infestation scores for the plants treated with these two compounds should be compared with the data in the second column, which lists similar data that were also taken on October 19, 1960, but from plants that had received only the preplant treatments as applied in May of 1959.

When the degree of infestation with the preplant treatments only was compared with that in the rows that were sidedressed with EN-18133, it was evident that the latter gave little or no control of root knot over that afforded by the preplant treatment only. In fact, in October 1960 only where Nemagon had been applied as a preplant treatment (treatments 7 and 10) was infestation essentially less than in the untreated check. On the other hand, DBCP (Fumazone in this instance) gave a very considerable increase in root-knot control when added as a supplemental treatment to most of the materials that were applied originally as preplant treatments (Fig. 1).

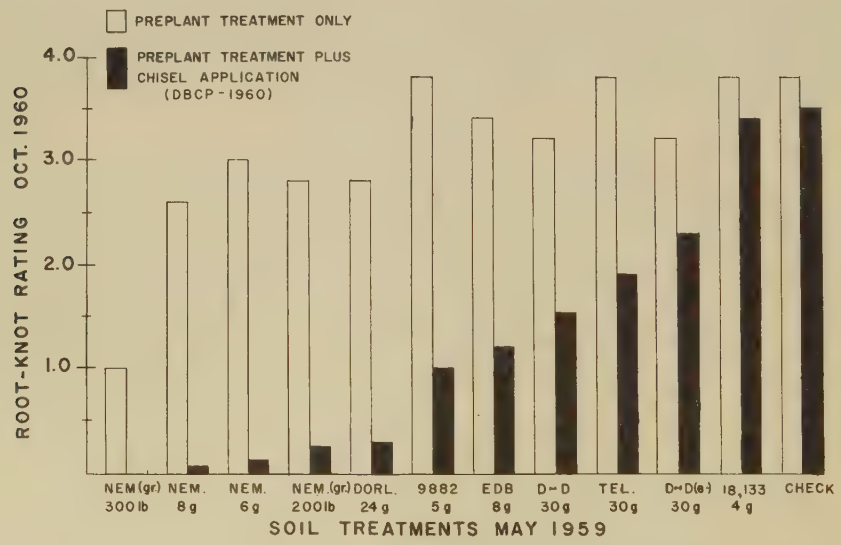


FIGURE 1. Increase in control of Meloidogyne hapla on Caryopteris when a supplemental treatment of DBCP was applied as a side-dressing in the nursery row 1 year after the application of various nematocides (see Table 1) as preplant treatments.

Wherever DBCP was used in both the preplant and supplemental treatments the final control was very good. Dorlone followed by DBCP also gave a low infestation rating. However, when the preplant treatment gave poor control, DBCP as a side-dress treatment sometimes gave very little additional control, as with EN-18133 in treatment 6.

Counts made of the number of M. hapla larvae in soil samples collected on October 18, 1960 showed them to be comparatively numerous in all plots except in the one treated in a preplant application with 300 pounds of Nemagon granules (treatment 10). The only other plot to show a lower larval count than the untreated check received EDB as a preplant treatment (No. 1). Why most of the treated plots showed a higher larval count than the check is not clear. The lesser plant growth in the check plot, which in turn furnished a smaller food source, may have been a partial cause of a lower nematode population in the fall of 1960.

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# A LONG TERM EXPERIMENT FOR PRESERVATION OF UREDIOSPORES OF PUCCINIA GRAMINIS TRITICI IN LIQUID NITROGEN

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The preservation of urediospores of Puccinia graminis Pers. f. sp. tritici Eriks. & E. Henn. in a viable condition for long periods would be desirable. Sharp and Smith (2, 3) developed a successful method for long-time preservation of urediospores in which the spores are first vacuum dried and sealed in glass tubes under vacuum and then stored in a refrigerator. Urediospores of P. graminis tritici have been kept viable for over 5 years by this method, but germination after that time is low. The method results in initial reduction of viability, which is more noticeable with some cultures than with others. Sharp and Smith's method has become a fairly standard and useful technique in most laboratories where work is being conducted with the cereal rusts.

Several attempts to quick-freeze urediospores of P. graminis tritici at -40° C have resulted in complete killing of the spores. H. T. Meryman (1) suggested the value of ultra-low temperatures for long-term preservation of biological materials. Liquid nitrogen (temperature -196° C) offers a means for freezing such materials to temperatures sufficiently low, theoretically, to halt decay or other changes during storage. The possibility occurred that freezing urediospores of P. graminis tritici in liquid nitrogen might successfully preserve them. The facilities of the American Type Culture Collection were used for a preliminary test. About 1 mg of urediospores was placed in each of six glass vials. Two vials were vacuum dried for 3 hours at .05 mm Hg and sealed under vacuum; two vials were sealed under vacuum at .1 mm of Hg; and two vials were sealed without drying or vacuum. One set of three vials was placed in a refrigerator at 4° C and the other set of three vials was frozen in liquid nitrogen. The latter was done by immersing the sealed vials directly in liquid nitrogen (-196°) for 20 minutes and then removing to a liquid-nitrogen refrigerator (-160° to -196°). Two days later the vials were removed from the refrigerator and defrosted by placing in a 37° water-bath for 4 minutes. The urediospores from all six vials were used to inject hypodermically 6- to 8-week-old plants of Little Club wheat and inoculate seedling leaves of the same variety by standard methods. The results of this preliminary experiment indicated that freezing in liquid nitrogen did not noticeably affect the ability of the spores to cause infection.

On the basis of these results a study was started to determine: 1) how long urediospores of P. graminis tritici can be kept viable when stored in liquid nitrogen; 2) what effect pretreatment has on storage; and 3) what differences, if any, there are in the effect of liquid-nitrogen storage on different rust races. The study is planned to cover a period of about 20 years, and the experimental plan is recorded below as a guide for future tests of viability.

Races 15B and 56 of P. graminis tritici were used. The cultures are numbered 15B-51A and 56-51A. They have been used in testing and research work in the cereals greenhouse at the Plant Industry Station, Beltsville, Maryland, since 1950. Their origin is unknown. Germination of fresh spores of these cultures is about 90%. Vacuum drying reduces germination of spores of 15B-51A to about 15% and of 56-51A to about 60%. Urediospores for the study were produced on 6- to 8-week-old plants of Little Club wheat that had been hypodermically injected with a urediospore suspension of each culture. On the afternoon of November 30, 1960, spores of each race were collected, placed in open Petri plates, and left in the laboratory to air-dry until the next morning. Samples of both races were used to inoculate differential wheat varieties and found to be highly infective and pure.

Four treatments were prepared on December 1, 1960, as follows:

VR - Vacuum dried for storage in a refrigerator. Spores for this treatment were vacuum dried at .05 mm Hg for 3 hours and sealed under vacuum in glass vials.

VF - Vacuum dried for freezing in liquid nitrogen. Spores for this treatment were vacuum dried and sealed as for VR.

A - Air dried. Air-dried spores were sealed at atmospheric pressure.

H - Hydrated. Air-dried spores were placed in a chamber over water for 4 hours and then sealed at atmospheric pressure.

Seventeen 7-mm glass vials, each containing about 2 mg spores, were prepared for each culture-treatment. The vials for the VR treatment were placed in an ordinary refrigerator (4° C). The vials for the VF, A, and H treatments were frozen by immersing in liquid nitro-

[1]Plant Pathologist, Collaborator, and Plant Pathologist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and Director, American Type Culture Collection.

gen and then placed in a liquid-nitrogen refrigerator (-196°).

On December 5, 1960, one vial of spores of each culture-treatment was removed from storage, and the spores from the vials were used to inject 6- to 8-week-old Little Club wheat plants. Subsequent development of rust indicated little difference between the races or treatments, though the spores from the A and H treatments appeared to cause more infections. The same procedure was used on January 10, 1961, with approximately the same results except that spores of 15B-51A that had been vacuum dried (VR and VF treatments) caused fewer infections. Tentative plans call for tests on viability 6 months and 1 year after storage and then at 1- to 2-year intervals.

In future tests of viability it is planned to use the spores to make germination tests on water agar, to inoculate seedling Little Club plants by standard techniques, and to inject 6- to 8-week-old Little Club plants. The first two methods will be used to determine percentage of viability of the spores while the third method will detect very low viability by the best test known at present.

The vials for future tests will be stored at the American Type Culture Collection in Washington, D. C., present address 2112 M Street, N.W., Washington 7, D. C.

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# SYMPTOMS IN RELATION TO INFECTION PATTERN IN WHITE OAK

John S. Boyce, Jr.[1]

Stambaugh[2] in Pennsylvania reported observations about the incidence of wilt in white oaks. He sampled all white and chestnut oaks near previously infected red oaks at 26 infection centers. Twenty-two of these trees at 13 centers were found to contain the wilt fungus. Half of them did not show any foliar symptoms although 8 of these 11 trees had excessive branch dieback. Three were non-symptom trees and could not have been detected on the basis of symptoms.

Although surveys for oak wilt have been made by the Southeastern Forest Experiment Station each year since 1950, only a few infected white oaks have been found. This may be due partly to the difficulty of recognizing symptoms in trees of the white oak group as opposed to red oaks.

Accordingly, the occurrence of crown symptoms in infected white oaks was recorded in as many trees as possible in order to rate their usefulness. Some trees were also cultured systematically to determine how readily the wilt fungus could be recovered from them. During the summers of 1956-1958, 10 infected white and post oaks (Quercus alba and Q. stellata) were detected at known infection centers in western North Carolina and eastern Tennessee. They were originally sampled for culturing by field crews because they had foliage abnormalities or branch dieback that was not attributable to some cause other than wilt.

Each tree was examined by the writer as soon as possible after infection was culturally confirmed. The color and distribution of abnormal leaves in the crown, branch dieback, and defoliation were noted. For systematic culturing, one 6-inch section was cut from each of three apparently healthy and three living diseased branches. In addition the stem was sampled at breast height at three equidistant points on the circumference, using an increment hammer.

Five chips from each branch section were placed on Barnett's oak wilt agar[3] in Petri dishes, and two or three increment cores from each stem point were placed in liquid Barnett's medium in test tubes. Cultures were incubated at 70° to 75° F for as long as 14 days to permit identification of the wilt fungus, Ceratocystis fagacearum (Bretz) Hunt.

Table 1 shows the incidence of symptoms in the infected trees. No one symptom occurred in all of the trees, but branch dieback and "water-soaked" leaves were noted in eight of the ten trees. Numerous dead leaves were recorded in seven trees and some had also shed leaves. Because only five trees showed varying amounts of defoliation, this was not a good field symptom of wilt in these white oaks.

Table 1. Crown symptoms associated with wilt infection in white and post oaks.

| Tree d.b.h. (inches) | Oak species | Crown symptoms: water-soaking of leaves | dead attached leaves | branch dieback | defoliation |
|---|---|---|---|---|---|
| 4 | white | + | + | 0 | + |
| 4 | white | + | + | 0 | 0 |
| 4 | white | + | + | + | + |
| 5 | white | + | + | + | 0 |
| 7 | white | + | 0 | + | 0 |
| 15 | white | 0 | 0 | + | 0 |
| 18 | white | + | + | + | + |
| 10 | post | + | 0 | + | 0 |
| 14 | post | 0 | + | + | + |
| 17 | post | + | + | + | + |
| % of trees showing given symptom | | 80 | 70 | 80 | 50 |

[1] Plant Pathologist, United States Department of Agriculture, Forest Service, Southeastern Forest Experiment Station, Asheville, North Carolina.

[2] Stambaugh, W. J. 1960. Investigation of oak wilt incidence in the white oak group. 2 pp. Processed.

[3] Barnett, H. L. 1953. Isolation and identification of the oak wilt fungus. West Virginia University Agr. Exp. Sta. Bull. 359T, 15. pp.

Four of seven trees yielded the wilt fungus from only one of three diseased branches (Table 2). It was isolated from two of three apparently normal branches of one tree, but not from symptom-free branches of other trees. Stem sampling with an increment hammer was relatively unreliable because four of the trees were negative on this basis.

Table 2. Isolation of wilt fungus from white and post oaks.

| Tree d.b.h. (inches) | Oak species | No. diseased branches: total sampled | No. diseased branches: with fungus | No. healthy branches: total sampled | No. healthy branches: with fungus | No. stem culture points: total | No. stem culture points: with fungus |
|---|---|---|---|---|---|---|---|
| 4 | white | 3 | 1 | 3 | 0 | 3 | 0 |
| 4 | white | 3 | 2 | 3 | 0 | 3 | 1 |
| 5 | white | 3 | 2 | 3 | 0 | 3 | 0 |
| 7 | white | 3 | 3 | 3 | 2 | 3 | 0 |
| 15 | white | 3 | 1 | 3 | 0 | 3 | 1 |
| 18 | white | 3 | 1 | 3 | 0 | 3 | 2 |
| 10 | post | 3 | 1 | 3 | 0 | 3 | 0 |

This small study showed branch dieback and water-soaked leaves to be good field symptoms of wilt in these white oaks. It showed also that the wilt fungus was not uniformly distributed in some of the trees, making cultural confirmation of infection more difficult than is usually the case in red oaks.

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# RACE 33 OF UROMYCES PHASEOLI VAR. TYPICA ARTH., A DISTINCT PHYSIOLOGIC RACE OF BEAN RUST FROM OREGON[1]

H. R. Hikida[2]

In 1952 Fisher (1) reported the reactions of 30 races of the bean rust, Uromyces phaseoli var. typica Arth., on differential bean varieties. Subsequently, in 1954, Sappenfield (2) described race 31, and in 1960 Zaumeyer (3) described race 32. These 32 races, designated numerically, comprise the known physiologic races of bean rust.

Bean rust has been observed in the Willamette valley of Oregon for many years. Fisher (1), in his study of the distribution of bean rust in the United States from 1941 to 1951, reported that races 18 and 22 in 1941 and race 29 in 1950 were collected in Oregon.

Table 1. Reaction of the differential bean varieties to several races of bean rust.

| Differential variety | Infection grade[a] produced by physiologic race | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Oregon | 3 | 4 | 8 | 15 | 17 | 24 | 27 | 29 |
| No. 643 | 2-3 | 2 | 1 | 1 | 5-6 | 0 | 3 | 1 | 0 |
| U.S. No. 3 | 10 | 9 | 3 | 5/3[b] | 10 | 10 | 3 | 2 | 8 |
| No. 650 | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 10 |
| No. 765 | 4 | 2 | 2 | 1 | 2 | 5 | 4 | 2 | 1 |
| No. 814 | 8 | 9 | 9 | 9 | 9 | 1 | 7 | 8 | 8 |
| No. 780 | 10 | 1 | 2 | 2 | 2 | 1 | 2 | 10 | 8 |
| Golden Gate Wax | 9 | - | - | - | - | - | - | - | 8 |
| No. 181 | - | 8 | 9 | 9 | 7 | 6 | 4 | - | - |
| Z-4 | - | - | - | - | - | - | - | 8 | 8 |

[a]Infection grades ranged from 0 for immunity to 10 for the highest degree of susceptibility.

[b]Numerator indicates size of pustule on upper leaf surface and denominator on lower surface.

During the period 1958 to 1960, 212 single spore isolates from 39 collections of bean rust made in the Willamette valley were inoculated onto the standard differential bean varieties. The results indicated the presence of a single race which was designated "Oregon." The reactions of this race on the differential varieties are compared in Table 1, with several previously described races selected on the basis of similarity in reaction on differential variety No. 643. The isolate differed from the selected races in reaction on several differential varieties. Since the Oregon isolate differed in reaction from all of the 32 previously described races, it is considered to be an unrecorded physiologic race of bean rust. This race is designated No. 33.

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# ARMILLARIA MELLEA ROOT ROT IN A NORTHERN WHITE PINE PLANTATION

S. B. Silverborg and R. L. Gilbertson[1]

Northern white pine (Pinus strobus) was recently found to be seriously damaged by Armillaria mellea (Fr.) Quél. in a 32-year-old 8-acre plantation at the Pack Demonstration Forest, Warrensburg, New York.

Previous reports of extensive damage by A. mellea in New York are limited to two natural stands of northern white pine, 10 and 22 years of age, in Clinton and Essex counties in northeastern New York (1, 2).

The Pack Forest plantation was planted in 1929 with a 6 by 6 foot spacing on Hinckley loamy sand soil. The average height and diameter in 1960 was 34.5 feet and 5.1 inches respectively. It was thinned once lightly in the spring of 1960. The presence of advanced symptoms of Armillaria root rot in July 1960 indicates that thinning was not a factor in the development of the disease.

To determine the extent of damage, a random one-tenth acre sample plot was established and all trees examined for the presence of the fungus and the associated rot. The extent of damage classified by diameter classes is summarized in Table 1. Of a total of 94 trees in the sample plot, 36 (38.3 percent) showed typical Armillaria root rot. No fruiting bodies or characteristic black rhizomorphs of the fungus were observed. The identity of the fungus therefore was confirmed culturally.

Table 1. Number of trees infected by Armillaria mellea in a one-tenth acre sample plot.

| DBH classes (inches) | Total number of trees | Number of trees | | % trees infected |
|---|---|---|---|---|
| | | infected | healthy | |
| 1.0-2.9 | 12 | 8 | 4 | 66.6 |
| 3.0-4.9 | 39 | 8 | 31 | 20.5 |
| 5.0-6.9 | 31 | 12 | 19 | 38.7 |
| 7.0-8.9 | 12 | 8 | 4 | 66.6 |
| Totals | 94 | 36 | 58 | 38.3 |

When the diseased trees were first observed in July 1960 only a few scattered trees had died as a result of A. mellea. However, many of the remaining diseased trees in the sample plot were in the advanced stages of the disease as evidenced by the presence of resin flow on the lower bole, a crustlike deposition of resin and needles at the root collar, and white mycelial fans in the roots and lower trunk extending to a height of approximately 6 to 14 inches. The fungus had completely encircled the trunk of many trees. However, crown symptoms were not generally apparent, suggesting a recent infection and an extremely rapid rate of growth of the pathogen.

Hinckley loamy sand on which the plantation was established is known to be characteristically low in soil nutrients. If A. mellea, as is generally believed, is able to infect only trees weakened by some other agency, apparently the disease is the result of planting white pine on soils deficient in nutrients. The resulting trees of low vigor are therefore susceptible to infection by A. mellea.

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# AN IMPROVED TECHNIQUE FOR TESTING EFFECTS OF SOLUBLE POSTHARVEST FUNGICIDES ON SPORE GERMINATION

George L. Greene[1]

The usual method for controlling postharvest diseases of various fruits utilizes a quick fungicidal dip. Because of possible damage that given toxicants may inflict, the period of contact of the fruit with the fungicidal agent must be rather short, but of sufficient length to kill all contaminating organisms. Consequently, when fungicides are being evaluated for dips, effective killing time as well as the most economical concentration must be determined for each compound. Such programs usually require a large number of time-consuming experiments.

In the past the centrifuge method as outlined by McCallan and Wellman (2) has been used to estimate spore-killing efficiency. However, as these authors state, this technique has several drawbacks. It requires the use of a large number of spores to compensate for losses in washing, and it is not very efficient for the study of short contact time periods. To overcome these difficulties a technique has been evolved utilizing filters of known pore diameter[2].

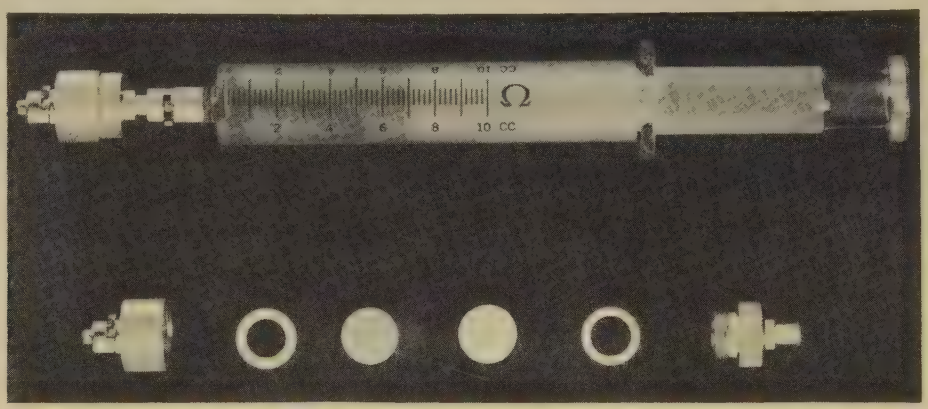


FIGURE 1. Upper--Locking-type syringe with Swinny adaptor in place. Lower--Details of adaptor showing casing, O-ring gaskets, filter disc and supporting metal screen.

The apparatus employed includes 10-ml syringes with metal locking-type tips and Swinny hypodermic adaptors[2] (Fig. 1) to hold the filter discs. The discs come in several known pore sizes, so a pore size smaller than the smallest diameter of the spore to be studied should be selected. However, as pore size is diminished the rate of flow also decreases. Experimentation has indicated that a 1.2 $\mu$ size is satisfactory for most spores and gives an adequate flow rate (1). A method similar to this has been used in the past with nematodes (3).

The procedure used is as follows: The concentration of spores is adjusted so that the final spore-fungicide mixture will contain the desired number of spores per ml. At zero time the spores and measured amount of fungicide solution are mixed thoroughly and drawn into the syringe. The Swinny tip is placed on the syringe and the liquid expelled. The tip is then transferred to a second syringe containing wash water which is expelled through the spores resting on the filter disc, thus removing the fungicide. The contact period can be considered ended as the washing process commences. After the first washing is completed the Swinny adaptor may be transferred to other water-filled syringes for further washings.

When the fungicide has been washed from the spores, a known volume of fresh water is drawn up through the Swinny tip into the syringe, thus suspending the spores at the desired concentration. The tip is then removed and drops of spore-water suspension expelled from the syringe onto clean glass slides which are placed in moist chambers and incubated at the optimum temperature for the specific pathogen. At the end of the desired incubation period spore germination counts are made.

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[2]Manufactured by the Millipore Filter Corporation, Bedford, Mass.

This method overcomes some of the difficulties encountered in the centrifuge technique (2). Since a filter is used, this technique would be of little value in assaying wettable powder toxicants. However, if the particle size of the wettable powder were small enough to pass through a filter that would retain the spores, then it is feasible that this modified technique could be extended to water-insoluble fungicides.

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## RHIZOCTONIA STEM CANKER OF TOMATOES

M. C. Strong[1]

In December 1960, a stem canker of greenhouse tomatoes was brought to my attention for diagnosis. The cankers had the brown, shrunken external appearance of Sclerotinia canker accompanied by wilting or death of the girdled tops. However, when cut open the hollow stem contained neither black sclerotia nor white mycelium but was filled with a coarse brown mycelium which was identified as Rhizoctonia solani Kuehn.

About 10% of the plants throughout the greenhouse range were infected with this canker. All the lesions appeared to have a pruning injury as the point of entry of the fungus, and all the cankers were at approximately the same height on the stem, suggesting a basidiospore infection by the Pellicularia filamentosa stage which took place during a relatively short time.

Subsequent inoculations with the Rhizoctonia isolate produced similar cankers. A wound of some sort was necessary for infection to take place.

Conover[2] reported a stem canker of field tomatoes caused by Rhizoctonia. In this case infection occurred through the mid-rib of leaves that were in contact with the soil. In the case of the greenhouse crop, the cankers were too far above the ground level for infection to have taken place in this manner.

Rhizoctonia has long been recognized as an important causal agent of damping-off and fruit rot of tomato but its potentialities as a canker disease may not have been realized.

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[2]Conover, Robert A. 1949. Rhizoctonia canker of tomato. Phytopathology 39: 950-951.

## STING NEMATODE IN ARKANSAS[1]

R. D. Riggs[2]

Sting nematode, Belonolaimus gracilis, was first described from Florida by Steiner in 1949 (3). The host was slash pine. Since then sting nematodes have been found in several States associated with various plants, and one new species, B. longicaudatus, described (2). These nematodes have been associated mainly with deep sandy soils. At the latest record, sting nematodes had been found in Alabama, Connecticut, Florida, Georgia, Louisiana, New Jersey, North Carolina, South Carolina, Texas, and Virginia (1). This report adds Arkansas to the States where this serious pest is known.

The Arkansas River Valley, near Van Buren, is comprised of various soil types, some of which are deep sandy soils. In the summer of 1960 a sample was taken from one such deep sandy soil known to contain root-knot nematodes. A small aliquot of the soil was screened and several males and females of sting nematodes were found.

Measurements of males and females were made and comparisons with the dimensions of the two described species indicated the specimens to be B. longicaudatus.

To determine the extent of the infestation, soil samples were taken in the general vicinity on similar soils where soybeans appeared unthrifty. Sting nematodes were recovered only from samples taken from the original infestation. No explanation was forthcoming as to the origin of this isolated infestation.

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## VERMICULITE MEDIA FOR GROWING FUNGI

Eugene H. Varney

Vermiculite saturated with a source of nutrients has been used in this laboratory for the past 3 years as a medium for fungi. It is especially good for infesting soil. Since no media with a vermiculite base were included in a recent methods manual[1], and since current research papers report the use of agar, corn meal sand, and whole grain media, a note on this use for vermiculite seems worthwhile.

Vermiculite (Terra-Lite) is easily poured or funneled into bottles or flasks and then saturated with any desired source of nutrients in water. About three parts of vermiculite to one part liquid will generally give the desired moisture content. V-8 juice, lima bean, and potato-dextrose extracts prepared by standard procedures were used extensively. One sterilization for about the same duration allowed agar media of comparable volume is sufficient.

Representatives of the genera Rhizoctonia, Verticillium, Fusarium, Cylindrocarpon, Pythium, Phytophthora, Stemphylium, Colletotrichum, Penicillium, and Aspergillus were among the fungi tested on vermiculite media. All grew rapidly and spore-formers fruited abundantly. Pythium sp. and Phytophthora fragariae produced zoospores when particles of mycelium-coated vermiculite were added to water. There was no evidence of any toxic effect on the zoospores. Verticillium produced both conidia and microsclerotia in abundance.

A vermiculite medium has a number of advantages over the commonly used media for growing soil fungi. Repeated and lengthy autoclaving is unnecessary, the medium is loose and well-aerated, and the mixture is easily broken into small particles which can be uniformly applied to the soil surface or incorporated into the soil. Unlike whole grain and other highly organic substrates or carriers, vermiculite appears to have no marked effect on the growth of secondary organisms which might affect disease development.

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[1]Johnson, L. F., E. A. Curl, J. H. Bond, and H. A. Fribourg. 1959. Methods for studying soil microflora-plant disease relationships. Burgess Publishing Co., Minneapolis. 178 pp.

## STEWART'S DISEASE: EXPECTED DEVELOPMENT ON CORN IN ILLINOIS IN 1961

G. H. Boewe

Stewart's disease, or bacterial wilt, of corn probably will be less destructive in Illinois in 1961 than in 1960. It probably will not occur as far north in the State as in 1960. This prediction is based on the close correlation that apparently exists between the winter temperatures and the amount of disease that develops during the following summer.

December temperatures were below normal throughout the State, ranging from 2 to 3 degrees below in the west and north and about 6 degrees below in the east and southeast. The coldest temperature in the United States on December 23, -25 degrees, was recorded at Joliet. It was -11 degrees at Urbana, the coldest December 23 at this place in 73 years of record. Heavy snowfall preceded the coldest weather of the month. All sections of the State, except the west and northwest, had above-normal snowfall; up to 6 inches above normal occurred in the east and southeast.

January temperatures averaged from 2 to 5 degrees below normal because of the unseasonably cold weather during the last 10 days of the month. Except in the extreme south of the State, there were 7 days with minimum temperatures of zero or below. The heaviest snowfall of the month occurred on the 19th, at the beginning of the coldest weather. This cold period continued into the first week of February. The rest of the month was warm and spring-like.

Winter indexes (sum of the mean temperature of December, January, and February) based on data from 91 weather-reporting stations in Illinois indicate that the early wilt phase of Stewart's disease on susceptible varieties of sweet corn will be destructive in the south one-fifth of the State. Across the State, directly north of this area, in a band about 80 miles wide, wilt is expected to be moderately severe. In the rest of the State it will probably be light to absent.

The late season leaf blight phase of Stewart's disease is expected to be light in the north half of the State and moderately severe to destructive in the south half.

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## BOOK REVIEW

"BACTERIAL PLANT PATHOGENS," by C. Stapp (translated by A. Schoenfeld), Oxford University Press, London. xviii + 292 pages. 1961. Price $6.75.

This nearly 300-page volume is written for the specialist -- or the specialist-to-be -- in bacterial plant diseases. Dr. Stapp, an outstanding authority in the field, offers the student a description of techniques and methods of identifying bacterial plant pathogens, and a thorough examination of 24 pathogenic species. Unfortunately for American readers, the author's list of 24 includes only those species found in Central Europe, those which he learned to known intimately as the former director of the Institute for Bacteriology and Serology of the "Biologische Bundesanstalt," Braunschweig, Germany.

Except for this limitation, Dr. Stapp's book has several features that recommend it. The introduction sets the stage for the reader, first, by giving perspective to bacterial plant pathogens in relation to bacterial pathogens of man and animals and to other causes of plant diseases; and second, by pointing up some of the common traits of bacterial plant pathogens.

In the first section of his book, the author discusses bacteriological technique, methods of identifying plant pathogens, including serological and bacteriophage methods, and the classification and nomenclature of the pathogens.

The second section, which constitutes more than 80 percent of the volume, is a detailed account of species beginning with Agrobacterium tumefaciens and ending with Xanthomonas pelargonii. Each account includes a description of the pathogen, symptoms, channel of infection and spreading in host, resistance, host plants, geographical distribution, and control. In all, this section includes five genera -- Agrobacterium (one species); Corynebacterium (five species); Erwinia (two species); Pseudomonas (10 species); and Xanthomonas (six species).

Nearly 100 excellent photographs illustrate the book. -- PAUL R. MILLER

## CORRECTIONS

REPORTER, December 1960 issue (Volume 44, Number 12). Pages 948-951.

The composition of the fumigant Trizone and the dosage used in my field plot tests of this material were stated incorrectly in my paper. I used Trizone in a solvent carrier formulation, but published the Trizone formulation that has no solvent carrier. The suggested corrections are as follows:

Table 1. (composition of Trizone) from 8% propargyl bromide; 31% chloropicrin; 61% methyl bromide to Trizone 3.7% propargyl bromide; 13.8% chloropicrin; 27.7% methyl bromide.

Table 3. Trizone rate/acre from 51 gallons to 461 pounds.

R. H. Converse

REPORTER, April issue (Volume 45, Number 4). On page 251, under the heading Pathogenicity Trials, paragraph 2, line 1, the second sentence should begin "The uninoculated" rather than "The inoculated." The Reporter regrets the error.